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THE NEW SYDENHAM
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VOLUME CXII.

HANDBOOK
OF
GEOGRAPHICAL AND HISTORICAL
PATHOLOGY.

BY
DR. AUGUST HIRSCH,
PROFESSOR OF MEDICINE IN THE UNIVERSITY OF BERLIN.

**Vol. II.—Chronic Infective, Toxic, Parasitic,
Septic and Constitutional Diseases.**

TRANSLATED FROM THE SECOND GERMAN EDITION

BY
CHARLES CREIGHTON, M.D.

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GEOGRAPHICAL AND HISTORICAL

PATHOLOGY.

THE ASSOCIATE EDITOR

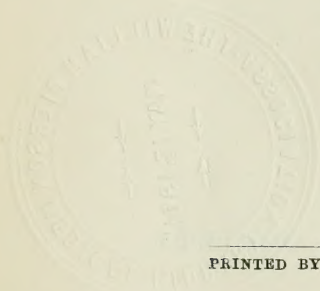
Professor of Medicine in the University of London

Volume - General and Special Pathology
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General and Special Pathology

General and Special Pathology



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GEOGRAPHICAL

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CHAPTER I.

LEPROSY.

§ 1. LEPROSY IN ANCIENT AND MEDIEVAL TIMES.

THE word "leprosy," with its colloquial equivalents¹ among the various nationalities of antiquity and the middle ages, was used during those periods in a collective sense, in the same way as the word "plague" was used; just as the latter included the most various diseases that occurred in epidemics and ran an acute course, so, under leprosy were embraced a number of chronic morbid processes, which had this much in common, that their most conspicuous symptoms were affections of the skin. And just as we are nowadays hardly able, with any certainty, to resolve into its elements the "plague" of antiquity and the middle ages, as described for us in the contemporary writings, and to decide

¹ Elephantiasis (of the Greeks), Lepra (of the Arabians), Morphaa (of the physicians of mediæval Christendom), Maalzey (the oldest German name). Of later designations in various countries, which are still current in part, I mention the following: Ladrerie (France), Aussatz (Germany), Melaatscheid (the Netherlands), Gafedad (Spain, where there is also the provincial name of Rosa Asturiensis), Gafeira (Portugal), Likthra (Iceland), Spedalskhed (Norway), Spetelska (Sweden), Kushta (India), Ngerengere (New Zealand), Morfea (Mexico), Mal rouge de Cayenne, Kakobe and Boasi (Surinam).

in each particular case whether it is really with bubo-plague that we have to do, or some other epidemic disease, and, if so, what other; we have a similar difficulty in extracting from the notices and sketches of "leprosy" that have come down to us from ancient and mediæval times any safe conclusion as to what is implied in each case under that very comprehensive term. This much, at least, we may infer from them, that venereal, scrofulous, and other skin troubles due to constitutional illness, had a place beside leprosy proper, as well as lupus, scabies, eczema, psoriasis, and other local cutaneous diseases. Keeping, then, that point of criticism in view, the history of leprosy in antiquity and the middle ages may be represented somewhat as follows.¹

The earliest accounts, that are at all reliable, of the occurrence of the disease on extra-European soil date from the time of the Exodus of the Israelites from Egypt, the wanderings in the Desert and the establishment of their power in Palestine. The inference that leprosy had been endemic in *Egypt* from the remotest times, would appear to be all the more warranted, inasmuch as all subsequent chroniclers speak of Egypt as the home of the disease, or, at any rate, as its headquarters.² An antiquity hardly inferior to this appears to belong to the leprosy of *India* and perhaps also to that of *China*; and, if we may discover no indication of it in the statements by Herodotus³ and Pausanias⁴ as to the *λευκή* of *Persia* in the sixth and fifth centuries B.C., yet, in *Japan*, according to the unanimous accounts of the chroniclers, it must have appeared as early as the thirteenth century B.C. (between 1234 and 1250).⁵

¹ For the history of leprosy, see the following: Raymond, 'Histoire de l'Elephantiasis,' &c., Lausanne, 1767; Hensler, 'Vom abendländischen Aussatze im Mittelalter,' Hamb., 1790 (1794); Danielssen et Boeck, 'Traité de la spedalskhed,' Paris, 1848; Munro, 'Edin. Med. Journ.,' Sept., Nov., 1876, and March, 1877; Häser, 'Lehrb. der Geschichte der Med. u. epid. Krankheiten,' 3rd. ed., Jena, 1876-82, iii, 70.

² See the remarks, in the sequel, on the history of leprosy as given by the Greek and Roman writers. Brugsch ('Histoire d'Egypte,' Leipzig, 1875, p. 42) has found, in the papyrus discovered by him, that "leprosy" is mentioned as early as the reign of Husapti, the 5th King of Egypt, having been prevalent, therefore, 2400 years before the Christian era.

³ *Ιστοριῶν*, i, 38, ed. Stein, Berl., 1856, i, 115.

⁴ *Βίοι παράλληλοι*, ed. Reiske, v, 490.

⁵ Schmid, 'New York Med. Rec.,' 1869, July, p. 194.

In several passages of the Bible,¹ mention is made, under the name "Sâraat," of a disease which was pre-eminently a skin disease, as appears from the minute account of the symptoms in Lev. xiii. In this passage it comes out in the clearest manner that the word "sâraat" is used to describe morbid states of the skin of various kinds, inasmuch as those affected with it were designated "clean" or "unclean" according to the type of the disease and the phenomena accompanying it. But that the word was used in a quite general sense is still more obvious from the fact that, in other passages,² "sâraat in the garment" is spoken of, and "sâraat in the walls of the house." The word "sâraat" means originally "stroke" (*Schlag*, Ger.), and in the passive sense also, "stricken," or "felled" (in Arabic the word for epilepsy or the "falling sickness" comes from the same root "saraa" = to throw to the ground). In its application in the passages above quoted, it corresponds clearly, therefore, to the German term *Ausschlag*, and the rendering of "sâraat" by *Aussatz* is justified only in the sense in which the latter name was given in former times to all kinds of "deposits" (*Nieder-schläge*), spots, and the like on the skin, the word *Aussatz*, in the sense of an eruption, surviving in the vernacular speech even to the present day in many parts of Lower Germany. That leprosy, in our sense of the word, is contained in this "sâraat," along with other diseases, is highly probable; but there can be even less doubt that the term comprehends many other skin diseases, such as psoriasis, scabies, and eczema, and perhaps also syphilitic affections.³

Reliable information as to the antiquity of leprosy in

¹ Lev. xiii, Numb. xii, 10, II. Kings, v, II. Chron. xxvi, 19.

² Lev. xiii, 47, and xiv, 39.

³ Finally ('Arch. für Dermatol.,' 1870, ii, 125) is of opinion that the passage in Leviticus where the description of "sâraat" occurs, is wrongly interpreted, inasmuch as the word "bâsar," which is used to signify the part of the body affected with "sâraat," is rendered by "skin" or "flesh," whereas it is employed there in a derivative sense, being a euphemism for "membrum virile." Whatever is said, accordingly, of the disease, has reference solely to "penis," and "sâraat" is no other than "syphilis." Seligmann, the historian of medicine, and a thorough master of the Semitic tongues, remarks on this conjecture ('Jahresber. über die Fortschritte in der Medicin,' 1870, i, 169): "Whether the dermatologists take Finally for a Hebraist, or the Hebraists look on him as a dermatologist, we have had no more remarkable piece of medico-biblical exegesis than this."

India is given in Wise's 'Commentary,'¹ where the lineaments of the disease, although obscured by other skin diseases, are still clearly recognisable. These commentaries are based upon the writings of Châraka and Sûsruta; so that we can trace leprosy in India back to the seventh century B.C. Indications of the disease, which has been known in India from time immemorial under the generic name of "kushta," had been given previously by Atreya, in whose Rig Veda Sanitâ of the fourteenth or fifteenth century B.C., there is mention made of it.² In *China* it was probably endemic, as Hobson³ thinks, among the original inhabitants of the country; but more definite information is wanting, and the case adduced by him, of a disciple of Confucius who became affected with "leprosy" (lae) is untrustworthy, inasmuch as the term "lae," as Hobson himself tells us, is used in Southern China, where true leprosy is most widely prevalent, in a quite general sense for scab. It is a noteworthy fact that the above-mentioned regions of the globe form one of the headquarters of leprosy at the present day.

At what time, in what regions, and under what circumstances the disease appeared first on the soil of *Europe*, cannot be made out with certainty from the scanty references to it that we meet with in the Greek writers of the pre-Christian era; and with all the less certainty for the reason that most of these accounts are already vitiated by the error of confounding leprosy with elephantiasis—a laxity of thought and speech which has obtained in all subsequent times down almost to our own day.

In the writings of the Hippocratic⁴ collection, there is mention of the "Phœnician disease" side by side with a malignant form of λέυκη (afterwards a term in general use for certain forms of leprosy), and obviously in connexion with the latter. According to Galen's explanation,⁵ this term corresponds to "elephantiasis;" but we are unfortunately again left in doubt whether by "elephantiasis" we are to understand leprosy, or elephantiasis in our sense of the word, inasmuch as

¹ 'Commentary on the Hindu System of Medicine,' Lond., 1860, p. 238.

² Munro, 'Ed. Med. Journ.,' 1876, Sept., p. 248.

³ 'Transact. of the China Branch of the Roy. Asiatic Soc.,' 1852, iii, p. 17.

⁴ 'Prorrhethikon,' lib. ii (at the end of the book), ed. Littré ix, 74.

⁵ Erotiani, 'Galen et Herodoti Glossaria in Hippocratem,' ex recensione Stephani, Lips., 1780.

Galen, in another passage,¹ uses the same word to denote pachydermia. It is equally uncertain what is meant by the "satyria" mentioned by Aristotle;² Rufus (*vide infra*) states that this word had been already used to designate leprosy, and Galen³ expresses the same opinion, again using, however, the ambiguous term "elephantiasis." Even if we discover leprosy in both of the diseases here spoken of, it would still remain a question whether the statements about them relate to Greece or to Egypt and Phœnicia; and the same doubt arises regarding the remark with which Rufus⁴ prefaces his own brief but unambiguous description of leprosy, that the disease, although it is a great and dangerous one and of frequent occurrence (μέγα καὶ χαλεπὸν νόσημα καὶ πολλοῖς γινόμενον), had been described hitherto only by Strato the Erisistratean (probably Strato of Lampsacus in the third century B.C.); for here also it is an open question whether this account of Strato, as well as the notice of the disease given by Rufus of Alexandria, does not relate rather to Egypt than to Greece.

Among the chroniclers both of the earlier and the later times of the Empire, there is complete agreement that the endemic leprosy of Egypt was not known to the older Greek physicians (*i.e.* to physicians practising in Greece), that the disease did not show itself in the Roman Empire until the last century B.C., and that it was a still longer time before it attained a general diffusion therein or among the neighbouring nationalities.

According to Lucretius,⁵

Est elephas morbus, qui propter flumina Nili
Gignitur Aegypto in medio, neque praeterea usquam.

Celsus, to whose trustworthiness no objection can be taken, says of it:⁶ "Ignotus paene in Italia . . . is morbus est, quem ἑλεφαντίασιν Graeci vocant." Plutarch⁷ makes the physician Artemidorus, a contemporary of Pompey, say that leprosy first became known in the Roman Empire in the time of Asclepiades (or in the last century B.C.). Caelius Aurelianus⁸ indicates Themison, the founder of the methodical school and the contemporary of Pliny, as the first Roman physician to describe the disease; and even in Galen⁹ we find the remark: "In

¹ 'Introductio, cap. xiii, ed. Kühn, xiv, 757, and, in the same sense, in 'Definitiones,' § 296 e. c. xix, 428.

² 'De generatione animal,' lib. iv, cap. iii, Opp. ed. Casauboni, Genév., 1605, i, 852.

³ 'De causis morbor,' cap. vii, e. c. vii, 29, and 'Lib. de tumoribus praeter naturam,' cap. xiv, e. c. vii, 727-28.

⁴ Oribasius, 'Collect. med.,' xlv, cap. 28, ed. Daremberg, iv, 63.

⁵ 'De rerum natura,' vi, 1114.

⁶ Lib. iii, cap. 25.

⁷ In 'Symposion,' vii, qu. ix, Opp. ed. Reiske, viii, 905.

⁸ 'Morb. Chron.,' lib. iv, cap. i, ed. Amstelod., 1755, 493.

⁹ 'Lib. ii, De method. med. ad Glauconem,' cap. xiii, e. c. xi, 142.

Alexandria plurimi elephantiasi . . . laborant. In Germania vero et Mysia rarissime affectus is grassari visus est. Et apud lactipotas Scythas nunquam fere apparet."

The period when leprosy began to be more widely diffused and more frequent in Europe may be inferred with a good deal of probability from the date of the making of laws and regulations by the authorities towards preventing and counter-acting the disease, which was generally considered to be a catching one; these consisted in part of laws regulating the marriage of leprous persons, and in part were directed to their segregation and reception into institutions for their care or cure (*leproseria, ladreria, meselleria*).

There exist legislative enactments on the marrying of lepers and on their segregation, made by Rothar, King of the Lombards in the seventh century,¹ by Pipin (757) and Charlemagne (789) for the empire of the Franks in the eighth century,² and for England in the year 950.³ The earliest account of the founding of leper-houses comes from the Frankish kingdom in the eighth and ninth centuries,⁴ from Ireland (Innisfallen), in the year 869,⁵ from Spain (Malaga in 1007,⁶ and Valencia in 1067),⁷ Italy and England (Chatham, Northampton, London, and other places)⁸ in the eleventh century, Sicily (Palermo),⁹ Scotland (Aldnestun, 1170), and the Netherlands (Ghent, 1147)¹⁰ in the twelfth century, and from Norway (Bergen, 1266)¹¹ and Switzerland (Zürich)¹² in the thirteenth century.

There is no doubt that the founding of the first houses for the reception of lepers did not take place until a period when the disease had already become spread to a consider-

¹ Lindenbrog, 'Codex legum antiquar.,' 1613, p. 609.

² Lobineau, 'Histoire de Bretagne,' Paris, 1707, i, 204.

³ Wharton, 'Anglia sacra,' ii, Praef. 32.

⁴ Virchow ('Archiv,' xviii (1860), pp. 138, 273, xix, p. 43, xx, pp. 166, 459), has published a very detailed and thorough investigation on the leper-houses in the Frankish kingdom and in Germany.

⁵ Belcher, 'Dubl. Quart. Journ. of Med. Sc.,' 1868, Aug., p. 38.

⁶ Martinez y Montes, 'Topogr. med. de la ciudad de Malaga,' Malaga, 1852, p. 504.

⁷ Morejon, 'Hist. bibliogr. de la med. Española,' i, 354.

⁸ For the history of leper-houses in Britain, see the admirable papers of Simpson, in the 'Edin. Med. and Surg. Journ.,' 1841, Oct., p. 301, 1842, Jan., p. 121, April, p. 394.

⁹ Profeta, 'La Sperimentale,' 1875, Sept., p. 294.

¹⁰ See the excellent account of the history of leprosy in the Netherlands by Israël, in 'Nederl. Tijdschr. voor Geneesk.,' 1857, i, 161.

¹¹ Bideknapp, 'Norsk. Mag. for Laegevidensk.,' 1860, xiv, 550.

¹² Meyer-Ahrens, 'Sweiz. Ztschr. für Natur- und Heilkunde,' 1841, vi, 302.

able extent. The majority of such houses appear to have been established between the eleventh and the thirteenth centuries; and they were founded in such numbers that, by the beginning of the thirteenth century, there appear to have been 2000 of them in France alone, and 19,000 in the whole of Christendom.¹ It would be a mistake, in my view, to infer from the multiplication of leper-houses, that there was a corresponding increase in the number of cases, or to take the number of the former as a measure of the extent to which leprosy was prevalent, or to conclude, as many have done, that the coincidence of the Crusades with that increased diffusion of the disease implies any intimate connexion between the two things, or that the rise in the number of cases was chiefly due to importation of leprosy into Europe from the East. In judging of all these matters, we must not leave out of sight the fact, already adverted to more than once, that the notion of "leprosy" was a very comprehensive one in the middle ages, not only among the laity but also among physicians, that syphilis was included therein as well as leprosy and a variety of chronic skin diseases, and that the diagnosis with a view to segregation of "lepers," or to their admission into leper-houses, was not made by the practitioners of medicine but mostly by the laity. One can readily understand that many a soldier from Europe, who set out for the East in good health, became leprosy in a country where want and hardships and a free kind of life all afforded opportunities for his acquiring the malady. But here again we shall have to take "leprosy" in its comprehensive sense; and, if the crusaders returning from the East brought back with them some disease which they communicated to people in Europe, it is much more reasonable to think of syphilis than of leprosy, inasmuch as the communicability of leprosy has not been proved hitherto by a single unambiguous fact. One thing of especial importance in the judging of these questions is that, when physicians came to be better acquainted

¹ Raymond, l. c., p. 106. Mezeray ('Histoire de France,' ii, 168) says of France in the 12th century: "Il y avait ni ville ni bourgade, que ne fust obligée de bâtir un hôpital pour les (lepreux) retirer;" and, for Italy, Muratori's statement ('Antiq. Ital. med. ævi,' iii, 53) is to the same effect: "Vix ulla erat civitas, quæ non aliquem locum leprosis destinatum haberet."

with the distinctive characters of syphilis at the time of its wide diffusion in Europe in the end of the fifteenth and beginning of the sixteenth century, the number of the leprous diminished considerably within a very short period; that fact is explained, not by any such sudden extinction of the disease, nor, as some have concluded, by the transformation of leprosy into syphilis, but according to the more natural assumption that a correct diagnosis of each disease had taught men to restrict the number of leprosy cases within proper limits.

When a revision was undertaken in France and Italy, at the beginning of the sixteenth century, of the overcrowded lazar-houses, the fact came out that in many of them by far the most of the inmates, and in some of them the whole, were suffering from various chronic skin eruptions, and that only a minority were suffering from true leprosy. Fracastoro,¹ for example, writing of Italy (Verona), says: “Et certe semper apud nos visus raro fuit is morbus, quamquam per civitates domus, quae hospitalia vocantur, et suppellectiles sumptibus publicis paratae structaeque videantur Elephantiacis suscipiendis. Verum quos ego haecenus vidi: nemo quidem aut pauci e suspectis Elephantiaci mihi visi sunt, sed leprosi [*i.e.* affected with scaly exanthems] solum, aut impetigine quadam fere detenti.”²

Still, we must recognise the fact that leprosy was prevalent in Europe as an endemic disease during the middle ages, that it gradually lost its importance as such from the sixteenth century onwards, and that there now remain to it in Europe only a few disease-centres, and these for the most part small, in which it still preserves its old endemic character.

This extinction of leprosy as an endemic took place, sometimes earlier, sometimes later in the various countries of Europe. In Italy a considerable remission of the disease was already noticeable towards the end of the fifteenth century; thus Beniveni³ says, when speaking of a case of leprosy observed by him in a foreigner: “Morbus qui in Italia”—he lived in Florence—“pene nunquam visus a medicis vix diagnoscitur.” During the sixteenth century leprosy disappeared almost entirely from that country, remaining confined to a few districts to be mentioned in the sequel.⁴ In the same period falls the general

¹ ‘De morbis contagiosis,’ lib. ii, cap. xiii, Opp. Venet., 1584, p. 946.

² See also Hensler, p. 232, and Simpson, l. c., 1842, Jan., p. 148.

³ ‘De abditis morbor. causis,’ cap. 98, in Dodonaeus, ‘Med. observ. rara exempla,’ Lugd. Batav., 1585, p. 241. See also the above-quoted passage from Fracastoro.

⁴ De Renzi, ‘Storia della med. in Italia,’ Napol., 1845, iii, 587.

decline of the disease in Spain,¹ where a few centres of it have maintained themselves down to the present time. In France also its subsidence began then, although it was observed at various places in considerable diffusion in the seventeenth and even down to the end of the eighteenth century. Thus, Simonin² states that leprosy was still common in Lorraine in the beginning of the seventeenth century; Briende,³ writing in 1787, says that it was still endemic in Upper Auvergne, especially in the district of Mont d'Or as far as the confines of Limousin, being known by the name of "mal s. main;" and Rochard⁴ points out that there were many lepers yet living at that time (1789) on the island of Belle-Isle-en-mer, which had served as a refuge for leprosy persons driven from the mainland, and that he had himself seen several at Rosalière. The extinction of leprosy falls at a somewhat later period in Switzerland, where numerous leper-houses were founded as late as the fifteenth century; later also in the countries of Northern Europe—in the Netherlands towards the middle of the seventeenth century,⁵ in Germany, where the disease, according to many observers, continued to be somewhat widely prevalent in the sixteenth century, in Denmark⁶ and in England and Scotland, from which last we have a notice of numerous cases of leprosy in Kingcase in the year 1693.⁷ From the Shetland Isles, we have the following by Edmondstone:⁸ "The session records mention that a day of public thanksgiving was observed in the ministry in the year 1742, when this disease [leprosy] was almost extinguished;" but he had himself seen several cases there at the beginning of the present century. On the Farøe Islands leprosy has completely disappeared since the middle of the eighteenth century.⁹

For the history of leprosy in *the countries of Nearer Asia* our information goes no farther back than the ninth and tenth centuries of the Christian era, the period from which date the writings of the so-called Arabian physicians Rhazes,¹⁰ Janus Damascenus,¹¹ Ali Abbas,¹² Avicenna¹³ and others, all

¹ Morejon, l. c. ² 'Recherch. topogr. et méd. sur Nancy,' 1854, p. 306.

³ 'Histoire de la Soc. de méd. de Paris,' 1787, v, mém., p. 311.

⁴ 'Journ. gén. de méd.,' 1789, lxxx, 365; Cabrol ('Rec. de mém. de méd. milit.,' 1848, ii, série vi, p. 51) makes the conjecture that the intractable skin diseases which are still met with on the island are the survivals of leprosy.

⁵ Israëls, l. c.

⁶ Danielssen et Boeck, l. c., p. 136.

⁷ Simpson, l. c., 1841, Oct., p. 328.

⁸ 'Edin. Med. and Surg. Journ.,' 1810, Jan., p. 162.

⁹ Manicus, 'Bibl. for Laeger,' 1824, i, 15.

¹⁰ 'Liber ad Almansorem,' v, cap. 32-35, Opp. Basil, 1544, p. 127.

¹¹ 'Practica,' tract v, cap. 3, 5, 14, Lugd., 1525, pp. 48, 49, 51.

¹² 'Disp. theor.,' i, cap. 74, viii, cap. 15, 16, 18; 'Pract.,' iv, cap. 3.

¹³ 'Canon,' lib. iv, Fen. iii, tract. iii, cap. 1, 2, 3, Fen. vii, tract. i, cap. 5.

of whom give definite information of the prevalence of the disease in Mesopotamia, Syria, and Persia. There is no medical history from those regions for the later mediæval period; but for the last three centuries the accounts of travellers, who had informed themselves as to the state of health and sickness there, are unanimous in declaring leprosy to be still endemic at many points. In the *Western Hemisphere*, according to the uniform statements of authorities in Guiana¹ and the West Indies,² leprosy was unknown until the arrival of the negro. For Bahia the earliest account of its occurrence goes no farther back than 1755. Also in Parana and Uruguay³ it is said to have become more widely spread in recent times, and in these countries also the outbreak of the disease is traced to the introduction of the negro race.⁴ The older observers considered it proved that the disease had been conveyed by contagion from the negroes to the natives of those countries; v. Leent, however, points out that these observations are plainly based upon a confounding of leprosy with syphilis, and that the conveyance of true leprosy from the one race to the other can only have taken place by way of intermarriage and inheritance. For Surinam the first information as to leprosy dates from 1728; in 1763 the number of lepers had increased so much that a leper-house had to be founded, and in 1812 they were counted at 500.

§ 2. PRESENT DISTRIBUTION OF LEPROSY.

The more recent history of leprosy, and its *geographical distribution at the present time*, tend to confirm the data, very

¹ Schilling, 'Diss. de lepra,' Traj. ad Rh., 1769; Bajon, 'Nachrichten zur Geschichte von Cayenne,' from the French, Erfurt, 1780, iii, 24; Campet, 'Traité prat. des maladies graves des pays chauds,' Par., 1802, p. 290; Bancroft, 'Natural History of Guiana,' p. 385; v. Leent, 'Arch. de méd. nav.,' 1880, Nov., p. 405.

² Pcyssonel, 'Philos. Trans.,' 1, part i, p. 38; Hillary, 'Observ. on the Changes of the Air, and the concom. Epid. Dis. in Barbadoes,' 2nd ed., Lond., 1766 (German transl., Leipzig, 1776, p. 385).

³ Wucherer, in 'Virch. Arch.,' 1861, xxii, 345.

⁴ Brunel, 'Observ. topogr. et méd. . . . faites dans la Rio de la Plata,' Par., 1842, p. 46.

defective and in part untrustworthy, which we possess as to the history of the disease in antiquity, the middle ages, and the modern period, and which point to Africa and Asia as its earliest and chief seats.

This holds good, first of all, for *Egypt*, where the medical accounts of the present and preceding centuries,¹ as well as the reports of all scientific travellers, represent the disease to be widely diffused as an endemic, equally throughout the whole basin of the Nile and on the shores of the Mediterranean and the Red Sea. To the same effect are the statements of all observers on the wide prevalence that leprosy has attained in *Abyssinia*,² both on the coast and in the plains and hill districts. Accounts to the same effect come from the coast territory of East Africa,—from *Zanzibar*³ and *Mozambique*,⁴ from *Madagascar*⁵ (both plains and mountains) and the adjoining small island of *St. Marie*,⁶—from *Mauritius*,⁷ where 150 patients had been admitted in 1874 into the leper-house near Port Louis,⁸ from *Reunion*,⁹ and from *St. Helena*.¹⁰ Among the North African coast states, Tripoli and Tunis are said to enjoy an immunity from leprosy, but the infor-

¹ See Prosper Alpinus, '*Medicina Ægyptiorum*,' Lugd. Batav., 1719, p. 56; Larrey, '*Mémoires de Chirurgie Militaire, et Campagnes*,' Paris, 1812-17, vol. i; Pruner, '*Krankheiten des Orients*,' p. 164; Griesinger, in '*Virch. Arch.*,' v (1853), p. 236; Vauvray, '*Arch. de méd. nav.*,' 1873, Sept., p. 161 (relating to Port Said).

² Combes et Tamisier, '*Voyage en Abyssinie*,' Paris, 1839, p. 280; Aubert-Roche, '*Annal. d'hyg.*,' 1846, xxxv, 5; Pruner, l. c.; Courbon, '*Observ. topogr. et méd.*,' &c., Par., 1861, p. 33; Blanc, '*Gaz. hebdom. de méd.*,' 1874, Feuille, p. 330 (met with leprosy west of Lake Tana).

³ Semanne, '*Essai d'une topogr. méd. de l'île de Zanzibar*,' Par., 1864, p. 36; Lostalot-Bachoué, '*Étude sur la constitution phys. et méd. de l'île de Zanzibar*,' Par., 1876, p. 48.

⁴ Roquette, '*Arch. de méd. nav.*,' 1868, Mar., p. 161.

⁵ Davidson, '*Edin. Med. Journ.*,' 1863, Mar., p. 832; Borchgrevink, '*Norsk. Magaz. for Lægevidensk.*,' 1872, iii, Raek. ii, p. 246.

⁶ Borius, '*Arch. de méd. nav.*,' 1870, Août, p. 81; he estimates the number of lepers on the island at 20 to 30, or 4 to 6 per 1000 inhabitants.

⁷ Kinnis, '*Edin. Med. and Surg. Journ.*,' 1824, Oct., p. 286; Lebonoté, '*Edin. Med. Journ.*,' 1877, Sept., p. 224.

⁸ '*Statist. Sanitätsbericht der kaiserl. deutschen Marine*,' 1874-5, p. 104.

⁹ Couzier, '*Journ. gén. de méd.*,' vii, p. 406; Allan, '*Monthly Journ. of Med.*,' 1841, Aug., p. 565; Pellissier, '*Considér. sur l'étiologie des maladies les plus communes à la Réunion*,' Paris, 1881, p. 49.

¹⁰ McRitchie, '*Transact. of the Calcutta Med. Soc.*,' 1836, viii, App. xxix.

mation¹ is of a scarcely trustworthy kind. In *Algiers*, at any rate, the disease is widely diffused (especially among the Kabyles);² and the same is true for *Morocco*,³ the *Canary Islands*,⁴ and *Madeira*.⁵ In the *Azores* it is said to be less frequently met with.⁶

According to Leared, there is in the vicinity of the city of *Morocco* a leper colony consisting of 200 persons, who are, however, in free communication with the whole neighbourhood. In the *Canary Islands* the first leper-house was founded in 1542, but the disease had probably existed there at an earlier date. The statement of Mendl that leprosy had decreased there in recent times in consequence of the improved wellbeing of the people, is not in agreement with the results (given by Friedel) of the official census taken in 1788, 1831, 1857, and 1860, which make the number of lepers then living to be respectively 195, 346, 500, and 600, so that we must suppose either the disease to have increased or the enumeration in later times to have been more carefully carried out. In *Madeira* the existing leper-house near Funchal, was founded in 1658; within the last thirty or forty years the disease has become rarer, most of the lepers being met with in a few districts on the west side of the island.

A very extensive region of leprosy is formed by the West Coast of Africa from Senegambia down to Cape Lopez. In *Senegambia* the disease is prevalent equally on the coast and in the more elevated regions of the interior;⁷ it is the same in *Sierra Leone*, where there were 103 lepers counted in 1860 among the 40,000 natives residing within the English colony.⁸ From the same region we have further accounts

¹ 'Report on Leprosy by the Coll. of Phys.,' Lond., 1867, p. 53.

² Baudouin, 'Gaz. méd. de Paris,' 1838, p. 771; Bertherand, 'Médecine et hygiène des Arabes,' Par., 1855; Guzon, 'Gaz. des hôpit.,' 1852, No. 27, p. 427; Bertrand, 'Rec. de mém. de méd. milit.,' 1867, Mars, p. 199 (who remarks that the disease known as "lèpre des Kabyles" is not leprosy but syphilis).

³ Jackson, 'Account of the Empire of Morocco,' Lond, 1814; Leared, 'Brit. Med. Journ.,' 1873, April, p. 404.

⁴ Friedel, in 'Virchow's Archiv,' xxii (1861), p. 340; Bolle, ib., p. 367; Mendl, 'Wien. med. Wochenschr.,' 1866, No. 35, p. 557.

⁵ Heineken, 'Edin. Med. and Surg. Journ.,' 1826, July, p. 15; Kinnis, ib., 1842, July, p. 1; Kämpfer, 'Hamb. Zeitschr. für Med.,' xxxiv, p. 161; Mendl, l. c.

⁶ Bullar, 'Boston Med. and Surg. Journ.,' 1840, xxvi, p. 135.

⁷ Thévenot, 'Traité des maladies . . . au Sénégal,' &c., Paris, 1840, 249; Chassianol, 'Arch. de méd. nav.,' 1865, Mai, 515; Borius, 'Considérations méd. sur le poste de Dagana,' Montp., 1864, and 'Arch. de méd. nav.,' 1882, Mai, 375.

⁸ Winterbottom, 'Acc. of the native Africans of Sierra Leone,' II, 113; 'Report on Leprosy,' 52.

for the *Gold Coast*,¹ for the *Benin Districts (Lagos)*,² for the *Niger*³ country, and for the *Gaboon (Cameroon Districts)*.⁴ It may be inferred from the statement of Daniell⁵ that leprosy occurs in these countries not merely on the coast, but as an endemic also in the interior; according to him leprosy persons are not unfrequently found among the slaves brought from the Soudan to the West Coast. On the *Loango Coast* (from Cape Lopez southwards) we come upon a territory free from leprosy;⁶ but the very defective medical information from these parts does not enable us to decide how far this immunity pertains to the most southern districts of the West Coast, to *Angola*, and the region of *the Congo*. It is certain that leprosy is endemic to a very considerable extent at the *Cape*,⁷ the fruitful districts on the east side forming an exception (Kretzschmar); *Natal*⁸ also is said to be free from the disease.

At the Cape there were in 1858 two leper asylums, or places of detention for lepers, the one being "Hemel-en-Aarde" (heaven and earth), a solitary place shut in by the mountains, distant a few days' journey from Cape Town and the coast; the other, Robben Island in Table Bay, thirty miles from the Herrnhut colony of Gnadenthal. According to the missionary Merensky,⁹ an endemic centre of leprosy has sprung up since 1850 among the Zulus who had migrated into Natal.

Besides Africa, the *continent of Asia with the archipelagos adjoining it* forms one of the headquarters of leprosy at the present day. This applies mostly to India and the eastern parts of Asia. On the other hand, in the countries of

¹ Clarke, 'Transact. of the Epidemiol. Soc.,' 1860, i, 106; Moriarty, 'Med. Times and Gaz.,' 1866, Dec., 663.

² 'Statist. Rep. of the Health of the Navy for 1864.'

³ Oldfield, 'London Med. and Surg. Journ.,' 1835, Nov., 403; McRitchie, 'Monthly Journ. of Med. Sc.,' 1852, May.

⁴ Ballay, 'L'Ogooué, Afrique équatoriale occidentale,' Paris, 1880, 40.

⁵ Daniell, 'Sketches of the med. topogr. . . . of the Gulf of Guinea,' Lond., 1849, 56.

⁶ Falkenstein, in 'Virchow's Arch.,' 1877, lxxi, 421.

⁷ Berneastle, 'Lancet,' 1851, Sept., 257; Kretzschmar, 'Südafrikanische Skizzen,' Leipz., 1853; Black, 'Edinb. Med. and Surg. Journ.,' 1853, April, 257; Scherzer, 'Ztschr. der Wiener Aerzte,' 1858, Nr. 11; Ref. in 'Lancet,' 1876, July, 32; Fritsch, in 'Virchow's Arch.,' 1865, xxxiii, 160 and 'Arch. für Anat. und Physiol.,' 1866, 733.

⁸ 'Report on Leprosy,' xi.

⁹ In 'Virchow's Archiv,' lxxxix (1882), p. 187.

Nearer Asia, so far as we can judge of their conditions of health from the available data, the disease has the character of a true endemic only within a few limited areas. Thus we have accounts of its occurrence at a few points on the southern coast of *Arabia* (particularly Muscat¹), and in the heart of the country;² further, in the mountainous districts of *Persia*³ and *Syria*,⁴ in *Cyprus*,⁵ and in some parts of *Turkestan* (especially Samarcand, Miankal, and Hissar⁶). In *Asia Minor*⁷ in recent times, only isolated cases have been observed—at Smyrna, in the neighbourhood of Sinope, and at other points on the shore of the Black Sea; in the Broussa district the disease appears to have become quite extinct, and it is said also that it no longer occurs in Trebizond.

In *Syria* leprosy is oftenest met with in the valleys of the Lebanon and Anti-Lebanon, mostly in small villages and seldom in the larger towns. The great number of lepers in Jerusalem, where there are several leper-houses, may be explained by their resorting thither from the country around. At Hebron, Nazareth, Safet, and Nablous, only occasional cases occur, just as at Beyrout, Jaffa, and other places on the coast, where leprosy is almost unknown. In *Cyprus* the disease is mostly found in the districts of Morfu, Lapethus, and Cythraea, situated on a damp level; the leper-house at Nikosia contained thirty-five patients in 1867. In *Persia* it is confined as an endemic to a few districts of the hill country of Irak Ajemi, in the provinces of Azerbijan and Khuzistan, particularly the localities of Chamsé, Kaswin, Sendjan, and Karadagh. It does not occur at all on the shores of the Caspian. The Persian lepers live together, outcast from society, in a most miserable state, in small and wretched colonies situated at some distance from the towns or villages.

¹ Lockwood, 'Amer. Journ. of Med. Sc.,' 1846, Jan., 82; Moore, 'Assoc. Med. Journ.,' 1856, Nov., 996.

² Palgrave, 'Journey through Arabia,' Lond., 1865, ii, 3.

³ Polak, 'Wien. med. Woch.,' 1855, Nr. 17, 'Wochenbl. der Ztschr. der Wiener Aerzte,' 1857, Nr. 47, 753, and in 'Virchow's Arch.,' 1863, xxvii, 175; Häntzsche, ib.; 'Report on Leprosy,' 71.

⁴ Pruner, l. c.; Tobler, 'Beitr. zur. med. Topogr. von Jerusalem,' Berl., 1855, 47; Robertson, 'Edinb. Med. and Surg. Journ.,' 1843, April, 246, 'Report on Leprosy,' xi, 54-56; 'Brit. Med. Journ.,' 1868, Sept., 341, and 'Wien. med. Woch.,' 1875, Nr. 13, 14; Langerhans, in 'Virchow's Arch.,' 1870, l, 453; Wortabet, 'Brit. and For. Med.-Chir. Review,' 1873, July, 173, and 'Med. Times and Gaz.,' 1880, Oct., 445.

⁵ 'Report on Leprosy,' 55.

⁶ Burnes, 'Transact. of the Calcutta Med. Soc.,' 1835, vii, 460.

⁷ Rigler, 'Die Türkei und deren Bewohner,' ii, 102; 'Report on Leprosy,' xiii, 60.

From none of the extra-European regions that are most afflicted with leprosy, do we obtain so complete information on the distribution of the disease and on its endemic extent within the several divisions of the country, as from *India* or that part of *India* which is under British rule. The exceedingly numerous accounts to hand,¹ which apply in part also to the Protected States and to the allied or Independent Native States, as well as to the French possession of Pondicherry, go to prove this much at the outset, that no part of that vast empire (including Ceylon), from Peshawur to Point-de-Galle, and from the Assam mountains to the Indus, is altogether free from leprosy; that there are, however, very considerable differences in the amount of it in various parts of the country, and that, in British India proper, there are three regions where the disease reaches a maximum—the hill country of Kumaon, the Province of Burdwan (Lower Bengal), and those parts of the Deccan and the Konkan that belong to the Bombay Presidency. According to the census of 1872, there were in the three Presidencies 99,073 lepers in a population of about 183 millions, or 5·4 per 10,000 inhabitants; of these 71,287 belonged to the Bengal Presidency with 136 millions (5·2 in 10,000), 13,944 to the Madras Presidency with 31 millions (4·4 in 10,000), and 13,842 to the Bombay Presidency with 16 millions (8·5 in 10,000). An analysis of these aggregates, carried out for the several provinces in each presidency, gives the following totals and the proportions per 10,000 inhabitants.

¹ The following are general references: Robinson, 'Med.-Chirug. Transact.,' 1819, x, part i, 27, 'Report on Leprosy,' xv—xix, 100—213; Lewis and Cunningham, 'Leprosy in India,' Calcutta, 1877. For Bengal, see: Macnamara, 'Leprosy,' Calcutta, 1866; Evans, 'Transact. of the Calcutta Med. Soc.,' 1829, iv, 245 (Tirhoot); Breton, *ib.*, 1826, ii, 245 (Chota Nagpore); Shortt, 'Ind. Annals of Med. Sc.,' 1858, July, 506; Richards, *ib.*, 1873, July, 303 (Orissa); Lewis and Cunningham, l. c., 14 (Kumaon), 'Report of the Governm. Charitable Dispens. established in Bengal and N. W. Provinces,' Calcutta, 1843, *passim*; Planck, 'Report on Leprosy in the N. W. Provinces,' Calcutta, 1876 (N. W. Provinces). For Madras, see: Van Someren, 'Med. Times and Gaz.,' 1874, April, 371 (District of Madras); Aubœuf, 'Contributions à l'étude de l'hyg. et des maladies dans l'Inde,' Par., 1862, 63 (Pondicherry); Day, 'Madras Quart. Journ. of Med. Sc.,' 1860, Oct., 286 (Cochin). For Bombay Pres., see: Vandyke Carter, 'Lancet,' 1872, Aug., 198, and 'Med.-Chir. Transact.,' 1873, lvi, 267 (general); Morehead, 'Clinical Researches,' ii, 664; Waring, 'Ind. Annals of Med. Sc.,' 1856, April, 506 (City of Bombay); Gibson, 'Transact. of the Bombay Med. and Phys. Soc.,' 1838, i, 66 (Gujerat); Don, *ib.*, 1840, iii, 4 (Sind).

Table of the number of Lepers in the various Provinces of India, with the proportion per 10,000 Inhabitants.

Bengal—		Madras—	
Bengal Proper	. 28,403=7·8	Littoral 9,872= 4·9
Behar 7,773=3·9	Inland Provinces .	. 4,072= 3·6
Orissa 1,077=2·4		
Chota Nagpore .	. 567=2·6	Bombay—	
Assam 309=1·6	Deccan 9,246=11·6
N. W. Provinces .	. 10,099=3·3	Konkan 2,753= 8·4
Oudh 7,831=7·0	Gujerat 1,534= 5·4
Berar 1,432=6·0	Sind 309= 1·4
Central Provinces .	. 2,807=3·0		
Punjaub 10,989=6·2		

These figures, it hardly needs to be said, afford no complete picture of the distribution of the sickness, inasmuch as they relate to very large territories, within which there are considerable differences for the several districts and even for the villages of a district. Carrying the analysis still further, we find that the following have the largest number of lepers, amounting to 20 or more per 10,000 inhabitants.

Table of the most leprous districts of India.

District.	Population.	No. of lepers.	Per 10,000 inhab.
Beerbhoon (Prov. Burdwan, Pres. Bengal).	695,921	2,872	41·2
Bancoora	526,772	1,578	30·0
Burdwan	2,034,745	4,604	22·6
Kumaon and Garhwal (Bengal) . . .	743,602	1,571	21·1
Banda (Prov. Allahabad, Pres. Bengal) .	108,771	214	19·6
Dehra Dhun (Prov. Meerut . . .)	115,771	220	19·0
Barsi (Prov. Sholapoor, Pres. Bombay) .	130,853	335	25·6
Sowda (Prov. Kandeish . . .)	124,519	312	25·0
Rajapoor (Prov. Rutnagherry . . .)	168,498	395	23·4

The disease is least frequent in the Madras Presidency, within which the Madras districts themselves are the worst, having 1 leper per 1000 inhabitants, and next to them Pondicherry, while, in the district of Coimbatore, the proportion falls to 0·2 per 1000.

On the extent of the sickness in the Protected and Independent States, we have only general data; the most con-

siderable leper-centres in these are on the plateau of Mysore (especially the district of Bangalore),¹ in the coast districts of Kattiwar,² in the State of Malwa,³ in Kashmir and Ladak,⁴ and in the State of Nepaul⁵ adjoining the severely affected Himalayan district of Kumaon.

We have no very particular facts as to the number of lepers in *Ceylon*; but, from the accounts to hand,⁶ we may conclude that the disease is common there also, chiefly on the southern and western coasts (provinces of Colombo, Galle, Matura, and Ballepittinge), less so in the interior and mountainous districts. From 1802 to 1862, 272 lepers had been admitted into the leper-house on the north bank of the Kalany some twenty miles above Colombo; during 1862 there were from 50 to 60 patients in it.

In *Lower India* we find a very considerable area of leprosy; we have accounts of its endemic occurrence from *British Burmah*,⁷ from the peninsula of *Malacca* (Penang, Singapore,⁸ Prince of Wales's Island), from *Siam*⁹ and from *Cochin China*.¹⁰ In British Burmah, according to the latest census,¹¹ the number of lepers was as follows:

Aracan, 125 in 484,362 inhabitants, or 3·8 per 10,000			
Pegu, 2072 in 1,662,658	„	or 12·4	„ „
Tenasserim 946 in 600,727	„	or 15·7	„ „

In the *East Indian Archipelago*, the most important leper-centres are on the west coast of *Java*, and in the mountainous districts, the disease being rare on its south and east coasts;¹²

¹ 'Report on Leprosy,' 188; van Someren, l. c.

² Vandyke Carter, 'Modern Indian Leprosy,' Bombay, 1876.

³ 'Report,' l. c.

⁴ Moorcroft, 'Travels.'

⁵ 'Report,' 191.

⁶ Pridham, 'Histor. and Statistical Account of Ceylon,' &c., Lond., 1849; Peacom, 'Edinb. Med. and Surg. Journ.,' 1840, Jan., 136; Kinnis, ib., 1842, July 6, October, 265; 'Report on Leprosy,' 90.

⁷ Dawson, 'Philad. Med. Examiner,' 1852, May; Richards, l. c.

⁸ 'Official Papers on the Med., Statist. and Topogr. of Malacca,' &c., Penang, 1830; Dick, 'Brit. Army Reports' for 1873, xv, 329; 'Report on Leprosy,' 197.

⁹ Friedel, in 'Virchow's Arch.,' 1863, xxvi, 183.

¹⁰ Sarrouille, 'Trois ans en Cochinchine,' Par., 1875, 17; Beaufils, 'Arch. de méd. nav.,' 1882, April, 279.

¹¹ According to Lewis and Cunningham, l. c., 9.

¹² Lesson, 'Voyage méd.,' 98; in 'Nederl. Tijdschr. voor Geneesk.,' 1858, ii, 223; 'Arch. de méd. nav.,' 1868, Sept., 165, 1869, Jan., 90.

other centres are in the *Andamans* and *Nicobars*,¹ in the elevated inland regions of *Sumatra* (especially *Paya-Combo*),² on the west coast of *Borneo* (particularly among the *Dyaks*),³ in *Celebes* (Province of *Menahasse*),⁴ in *Flores* and in the interior of *Timor*,⁵ in *Banda* and some other islands of the *Moluccas*,⁶ and in the *Philippines*.⁷ On the other hand, it is seen rarely on *Banka* (chiefly among the Chinese working in the mines),⁸ on *Amboina*,⁹ in the *Lampong* territories of *Sumatra*,¹⁰ or in the Government of *Macassar* (*Celebes*).¹¹

In the *Chinese Empire*,¹² the southern and eastern coast districts, as far up as the mouth of the *Yang-tsze-Kiang*, have been the headquarters of the disease from time immemorial. It is met with more rarely in the interior, and mostly towards the north of the empire. The provinces most afflicted by it are *Quang-Tung* (Canton), *Tu-Kiang*, and *Che-Kiang*, between the latitudes of 22° and 31° N. In *Pekin* it is seldom seen.

The number of lepers living in the Province of Canton is estimated at 10,000; in the villages round the city, there is 1 leper to every 1000 or 2000 of the population; and there is hardly a district in the whole province without them, although it is the coast districts that are mostly affected. In the neighbourhood of the City of Canton there are two leper villages, the one harbouring 700 to 800 and the other 1000; but most of their inhabitants are nothing more than the descendants of lepers.

In *Japan*¹³ leprosy is prevalent in widest diffusion, from the coasts far into the interior; only the *Loo-Choo Islands*, according to a credible report, enjoy an immunity. Here,

¹ Hodder, 'Brit. Army Reports' for 1875, xvii, 261.

² In 'Arch. de méd. nav.,' 1867, Oct., 248.

³ Ib., and 1872, Jan., 22.

⁴ Ib.

⁵ 'Archiv de méd. nav.,' 1867, Oct.

⁶ Heymann, 'Krankh. der Tropenländer, 204, 'Arch. de méd. nav.,' 11. cc.

⁷ Taulier, ib., 1877, Dec., 411.

⁸ Ib., 1873, Feb., 103.

⁹ v. Hattem, 'Nederl. Tijdschr. voor Geneesk.,' 1858, ii, 538.

¹⁰ L. c.

¹¹ In 'Arch. de méd. nav.,' 1871, April, 248.

¹² See Lockhart, 'Med.-Chir. Rev.,' 1842, July, 150, 'Monthly Journ. of Med. Sc.,' 1846, March, 164; Wilson, 'Med. Notes on China,' Lond., 1846; Hobson, l. c., 'Report on Leprosy,' 72-79; Shearer, 'Edinb. Med. Journ.,' 1872, Jan., 596; Wong in Leudesdorf's 'Nachrichten,' ix, 22.

¹³ Schmid, 'New York Med. Record,' 1869, July, 193; Wernich, 'Geogr.-med. Studien,' Berlin, 1878, 200.

as in all other countries, there are some points much more severely affected than others, such as the Bay of Nagasaki, the Bay of Yeddo, Miako, and Oruma. Wernich found almost the whole population leprous in a considerable village between Yeddo and Yokohama. Of the occurrence of the disease in *Siberia*, I have no information; in *Kamtschatka* it is said to be very often met with, but the statement¹ is not quite trustworthy, as there seems to have been some confounding of it with syphilis.

On the Continent of *Australia*, leprosy has been found hitherto only in occasional cases among the Chinese immigrants, and principally in the gold districts of Victoria (in the vicinity of Ballaarat, Castlemaine, and Beechworth).² It is quite unknown in South Australia, Western Australia, and *Tasmania*. On the other hand, it is widely prevalent among the natives of *New Zealand*, being known by the name of "Ngerengere." According to information dating from 1854,³ the disease had decreased very much in recent times—whether in proportion only to the frightful depopulation of the native territory or absolutely, is not stated—and it is now met with almost exclusively in the interior.

How far Brunet's statement is justified, that leprosy occurs on all the islands of *Oceania*,⁴ I am unable to decide in the absence of relevant information as to points of detail. In the *Society Islands*⁵ (Tahiti), the disease is known under the name of "Oovi," but it is rare. The *Hawaiian Islands* (Honolulu), are the only group from which we have accounts of leprosy spreading widely within quite recent times; but this intelligence, also, is wanting in trustworthiness.

In the small tract on leprosy by Macnamara (Calcutta, 1866), already quoted, a statement is given of a physician Hillebrand, practising at Honolulu, according to which leprosy was imported into the island in 1840 by Chinese, and rapidly attained so enormous a diffusion that, at

¹ 'Inosemzoff, 'Med. Ztg. Russl.,' 1844, Nr. 6.

² 'Report on Leprosy,' xiv, 80-82.

³ Thomson, 'Brit. and For. Med.-Chir. Rev.,' 1854, April.

⁴ 'La race Polynésienne,' Par., 1876, 44.

⁵ Hercouet, 'Étude sur les maladies des Européens aux îles Tahiti,' Par. 1880, 71.

the time of writing (1865), 3·5 per 1000 of the whole population were affected with it. The same story has been told subsequently by Kneeland,¹ and after him by Emerson,² who places the date of the importation, however, in 1856; he adds that there were 684 persons—Romanowski³ gives 806—living in the leper colony on Molokai. Milroy⁴ has thrown doubts—and, it seems to me, with good reason—on the statement of Hillebrand; and the matter in question is rendered still more doubtful by the remark of Enders,⁵ that many so-called cases of leprosy in Honolulu are to be counted as syphilis.

On the soil of *Europe*, leprosy occurs endemically at the present day only in small and, for the most part, closely circumscribed areas. It is still prevalent to a considerable extent in the *Iberian Peninsula*. In *Spain*, as far as I am able to judge from the very scanty, and in part somewhat antiquated, data before me,⁶ the headquarters of leprosy are the Provinces of Catalonia, Andalusia, Galicia, Asturia, and Granada, being coast territories; in *Portugal*, the Provinces of Beira, Estremadura, and Algarve. There are not many facts as to the number of lepers in the two kingdoms. In 1851, the official returns for Spain gave 284 lepers as living in nine provinces. In 1877 a new enumeration of the lepers was undertaken, which has proved, in the result, to be very imperfect; thus in Valencia, only 116 are returned, while it could be shown that many leprous persons there had kept themselves concealed; in Malaga, also, as Martinez y Montes states, the number of lepers is much greater than has come to official cognisance. In the Province of Alicante a new leper-house has been opened lately—a proof that the disease is still somewhat prevalent there. For Portugal, the number of lepers was given in 1821 at 800; in his report dating from 1838, Baptiste assigns the mountainous district of Lafoês (Prov. Beira), as the headquarters of the disease, the number of lepers in 477 communes being 3000 (which d'Almeida says should read 300).⁷

¹ 'Boston Med. and Surg. Journ.,' 1873, March, 233.

² 'Brit. Med. Journ.,' 1880, Sept., 401.

³ 'Arch. de méd. nav.,' 1881, Oct., 314.

⁴ 'Med. Times and Gaz.,' 1875, July, 66.

⁵ 'Brit. Med. Journ.,' 1876, Dec., 731.

⁶ See, for Spain: Soares, 'Jorn. da Soc. das sc. med. de Lisboa,' vi, 1; Borrow, 'Five Years in Spain,' Ger. Transl., Bresl., 1844, ii, 183; Webster, 'Med.-Chir. Transact.,' 1860, xliii, 27; Virchow, 'Arch.,' 1881, lxxxiv, 417.

⁷ For Portugal: Baptiste, 'Jorn. da Soc. das sc. med. de Lisboa,' 1838, May;

Two small centres of leprosy, on which a good deal of light has been thrown recently, are met with in *Italy*, the one on the Gulf of Genoa (*Riviera di Ponente*), the other on the Adriatic in the neighbourhood of the small town of *Comacchio* situated in the marshes of Ferrara. Quite lately attention has been directed to a somewhat more considerable endemic in *Sicily*, which has been increasing within the last thirty or forty years. As regards *Comacchio*, where the disease is said to have appeared first in 1806,¹ the number of lepers in the town amounted in 1845 only to about a dozen;² occasional cases from *Comacchio* were observed in Bologna as late as 1868,³ but the endemic in that quarter appears to be now extinct. The small endemic on the *Geonese Riviera* is clearly a survival of the great leprosy centre that once extended from Chiavari (on the Riviera di Levante), along the Italian and French coasts as far as the mouths of the Rhone, being represented in Provence down to the end of last century by the somewhat frequent cases in Martigues, Vitrolles, and a few localities in the neighbourhood of Marseilles and of Toulon,⁴ and on the Riviera di Levante as late as the first ten years of the present century, by the persistence of the disease in Chiavari, Varese, and other places. At all these points, as well as in the villages of Pigna, Castel Franco, La Turbie, and others belonging to the quondam Duchy of Nice, leprosy has died out entirely during recent years, and it is now confined to a few valleys opening towards the coast. An enumeration made in 1843 puts the number of lepers in the whole district at 100.⁵ In 1858 the Italian Government fitted up a monastery on the highest point of San Remo as a leper-house, into which forty

d'Almeida, *ib.*, Aug.; Soares, *l. c.*; Kessler, in 'Virchow's Arch.,' 1865, xxxii, 257; Virchow, *l. c.*; Peacock, 'Lancet,' 1870, Dec., 773.

¹ Parola, 'Saggio di climatol. e di geogr. nosol. dell' Italia,' Torino, 1881, 502.

² Medici, 'Annal. univ. di med.,' 1836, Sept.; Verga, 'Sulla lebbra,' Milano, 1846.

³ Sgarzi, 'Gaz. med. Lombard,' 1868, Nr. 11.

⁴ Vidal, 'Hist. de la Soc. de méd. de Paris,' 1779, i, Mém., 161; Valentin, 'Bull. de l'école de méd. de Paris, 1807, 48; Foderé, 'Journ. complém. du dictionn. des sc. méd.,' 1819, iv, 3; Fuchs, 'Diss. de lepra Arabum,' Wircob., 1831.

⁵ Trompeo, 'Giorn. delle sc. med. di Torino,' 1843, Gennajo; Boeck et Danielssen, *l. c.*, 185.

patients were received;¹ on visiting it in 1877, I found only six or seven lepers, and the doctor in charge informed me that no new cases had presented themselves for two years, so that we may look for a complete extinction of the endemic there also. The first beginnings of the recently developed endemic of leprosy in *Sicily*² go back to the end of the seventeenth century, when the disease showed itself in the village of Avola (Prov. Syracuse), which had been quite free from it up to that time; fifty years later, it appeared at Buccheri, Floridia, Solarino, and Naso (Prov. Messina); towards the end of the eighteenth century (1780 and 1790) at Trapani and on the adjoining island of Favignana; in the early years of this century at Cefalù (Prov. Palermo) and Lipari (Prov. Messina), in 1830 at Monte San Giuliano and at Mirto (Prov. Trapani), in 1854 at Carini (Prov. Palermo); while, finally, from 1860 to 1870, occasional cases have occurred at Petralia, Girgenti, Polizzi, and Sciacca.

Of 114 cases observed lately, and all well authenticated, 75 came from Avola, 10 from Floridia, 9 from Trapani, 8 each from Palermo, Solarino, and Naso, 7 each from Castellamare, Buccheri and Lipari, 6 each from Cefalù and Monte San Giuliano, 5 from Favignana, 3 from Mirto, and 1 each from Carini, Petralia, Polizzi, Girgenti, and Sciacca; so that while there were 2 lepers to every 9000 inhabitants of the coast, there were 5 to the same number of residents inland.

Only a few general references to the endemic occurrence of leprosy in the *Balkan Peninsula* are to hand from recent times. According to these, there are still existing small centres of the disease on the coast of the Ejalet of Salonica (Thessaly and Macedonia); in Constantinople cases occur very seldom, and the leper-house in the Scutari suburb is mostly used for leprosy patients coming from Asia Minor. In Monastir, Janina, and many other places, leprosy is now quite unknown.³

An enumeration of lepers in *Greece*⁴ in 1851 puts them at 350,⁵ but there are unfortunately no precise facts as to their

¹ Thaon, 'Nice médical,' 1876, Nr. 3; Köbner, 'Viertelj. für Dermatologie,' 1876, iii, 3.

² Profeta, 'Lo Sperimentale,' 1875, Sept., 294; Ferrari, 'La lebbra in Sicilia,' Catania, 1878.

³ 'Report on Leprosy,' xiii, 68.

⁴ Rigler, l. c., ii, 114.

⁵ Ibid.

distribution in various parts of the country. More recent information¹ indicates that the endemic centres of the disease occur mostly in the eastern districts, particularly in a few villages in the neighbourhood of Parnassus. On the *Ionian Islands* leprosy would appear to be now rare (eighteen lepers were counted in Corfu in 1862);² on the other hand, the disease is prevalent as an endemic to a greater or less extent on many *Islands of the Ægean*, such as *Samos*,³ *Rhodes*⁴ (with the small islands around it), where there were some 300 lepers living in 1862, *Chios*⁵ and *Mytilene*, where there is a village forming a completely isolated leper colony, although many lepers travel the island as beggars in the fine weather.⁶ But the chief seat of leprosy in this region is *Crete*; an enumeration made there in 1833 of persons notoriously leprous gave a total of 628, of whom 522 were in the province of Candia, 64 in Retino, and 42 in Canea; but many more cases, who had only lately become leprous, or who had been hidden by their relatives, remained uncounted; and we shall not go far wrong in following Smart,⁷ and placing the number of lepers living on the island, among a population of 250,000, at 900, giving a ratio of 3·6 in the 1000. This enormous prevalence of leprosy in Crete has lately been confirmed by Brunelli.⁸

In *Roumania*⁹ and in *Hungary*,¹⁰ as well as at many other parts of Europe, occasional cases of leprosy occur; but in those countries the disease has long since ceased to be endemic. In the region of *Southern Russia* also,—which had been much afflicted with leprosy even as late as the first quarter of this century,—extending from the *Crimea*, along the Sea of Azov and the *Caucasian Frontier* as far as the Ural Steppe,¹¹ the

¹ Dekigalla, 'Gaz. hebd. de méd.,' 1860, 108; Cigalla, 'Annal. univ. di med.,' 1865, Gennajo 3; Ornstein, 'Journ. des conaiss. méd.-chir.,' 1866, 367.

² 'Report on Leprosy,' xiii, 65.

³ Mengozzi, 'Gaz. méd. d'Orient,' 1861, April.

⁴ 'Report,' xii, 58.

⁵ Pasqua, 'Bull. gén. de therap.,' 1880, 15, Dec., 507.

⁶ Bargigli, 'l'Union méd.,' 1878, Nr. 49, 633.

⁷ 'Med. Times and Gaz.,' 1853, Oct., 444.

⁸ 'Annali univ. di med.,' 1866, Dec., 461, 1867, Gennajo 3.

⁹ Scheiber, 'Viertelj. für Dermatol.,' 1875, i, 363.

¹⁰ Schwimmer, 'Pester med.-chir. Presse,' 1880.

¹¹ See Martius, 'Abhandl. über die krimmische Krankheit,' &c., Freib.,

disease appears to have decreased materially of late.¹ The same applies to the *Baltic Provinces* in recent times.²

In a fishing village at the mouth of the Danube, inhabited by Russians and Greeks, a small centre of leprosy has developed within the last twenty years,—it is said in consequence of importation.³ In the *Caucasus* the disease still occurs in a few Cossack villages ("stanitzas" or fortified stations); Popoff⁴ refers to a leper-house opened since 1850 in a Cossack village in the Government of Stavropol, and Liebau⁵ found another in a "stanitzza" on the Terek, in which, however, there were only three patients.

In *Sweden*, where many cases of leprosy were met with at the end of last century and beginning of this,⁶ in the districts of Angermanland, Medelpad, Helsingland, Upland, and Bohus, a considerable decrease of the disease has been observed of recent years, as will appear from the following statistics:⁷

Table of Lepers in Sweden from 1867 to 1879.

District.	1867	-68	-69	-70	1871	-73	-74	-75	-76	-77	-78	-79
Gefleborg . .	63	58	77	85	86	103	94	91	89	90	83	86
Jemtland . .	1	1	1	1	1	2	2	—	—	—	—	1
Wester-Norrland	3	3	3	3	3	3	3	6	6	6	7	7
Kopperborg .	10	9	12	9	8	10	9	9	9	2	2	4
Blekinge . .	1	1	1	1	1	1	1	1	—	—	—	—
Gottland . .	—	1	1	1	1	1	1	—	1	1	1	1
Upsala . .	—	—	—	—	—	—	—	—	3	2	2	1
Wermland .	—	—	—	—	—	—	—	—	—	—	1	—

1819; Plachoff, 'Von dem tuberculösen Aussatz der donischen Kosaken u. s. w.,' Moskau, 1842 (in Russian); Krebel, 'Med. Ztg. Russl.,' 1846, Nr. 38.

¹ The most recent accounts of leprosy in Astrakhan by Oldekop ('Virchow's Archiv,' xxvi (1863), p. 106), and Meyerson (ib., xxxi (1865), p. 446) are based on only a few observed facts. When I was in Astrakhan in the spring of 1879, I got sight of only a single case, and that a doubtful one, notwithstanding every inquiry. In reply to my questions, the practitioners there all assured me that the disease is now very rare.

² Wachsmuth, 'Arch. für klin. Med.,' 1867, iii, 1; Bergmann, 'Die Lepra in Livland,' St. Petersburg, 1870.

³ Vignard, 'Gaz. méd. de Paris,' 1877, 563.

⁴ 'Med. Ztg. Russl.,' 1854, 381.

⁵ Liebau, 'Petersb. med. Ztschr.,' 1866, xi, 284.

⁶ Huss, 'Om Sverges endem. sjukdomar,' Stockh., 1852, 10, 11, 14, 34, 43.

⁷ I have taken the data from the 'Sundhets-Collegii Berättelse' for the respective years.

From this it appears that during the past twenty years there has existed only a small endemic centre in Gefleborgslän; of the 86 cases living there in 1879, 17 belonged to Ljudal, 18 to Gerfsö, 15 to Delsbo, and 7 to Alfta, the remainder occurring singly here and there.

One of the most considerable leprosy-regions of Europe is the west coast of *Norway*, from Stavanger up to Tromsöe, most of the cases belonging to the departments of Søndre and Nordre Berghus, which have been the headquarters of the disease in Norway from the first.¹

Since 1856, a careful reckoning of the numbers of lepers in Norway has been made every year, from which the satisfactory conclusion is drawn that the number of cases has decreased nearly by one half in nineteen years, having fallen steadily from 1859. The figures are as follows :

Table of the number of Lepers in Norway from 1856 to 1874.

1856 ... 2,847	1863 ... 2,660	1869 ... 2,276
1857 ... 2,773	1864 ... 2,639	1870 ... 2,050
1858 ... 2,774	1865 ... 2,603	1871 ... 1,987
1859 ... 2,785	1866 ... 2,563	1872 ... 1,943
1860 ... 2,741	1867 ... 2,497	1873 ... 1,874
1861 ... 2,717	1868 ... 2,413	1874 ... 1,832
1862 ... 2,685		

The following table gives a summary of the number of cases in the various departments of the country, and shows

¹ Among older authorities, see : Pontoppidan, 'Versuch einer natürlichen Historie von Norwegen,' Kopenh., 1754, ii, 480; Martin, 'Abhandl. der Schwed. Akad. der Wiss,' xxii, 301; Ström, 'Phys. oecon. og med.-chir. Bibl.,' 1795, July, 223; Pfefferkorn, 'Ueber die Norweg. Radesye und Spedalskhed,' Altona, 1797. Recent authorities: Horn, 'Norsk. Mag.,' 1841, ii, 42; Boeck, ib., 1842, iv, 1, 127; Danielssen, ib., v, 131; Steffens, ib., 1843, vi, 229; Hoffmann, ib., 1846, ix, 251; Boeck et Danielssen, l. c.; Hjort, 'Norsk. Mag.,' 1856, N. R., x, 649; 'Discussion i det Norske med. Selske angaaende spedalskheden,' Christ., 1857; Holmsen, 'Norsk. Mag.,' 1857, ix, 129; Bidentkap, ib., 1858, xii, 398, 1860, xiv, 535, 713, 809, 889; Lochmann, ib., 1871, Tr. R., i, 129; Hjort, 'Om spedalskheden i Norge,' Christ., 1871, and 'Norsk. Mag.,' 1872, ii, 105; Buchholz, 'Om spedalskheden som folkesygdom,' Christ., 1872; Hansen, 'Norsk. Mag.,' 1872, ii, 1, and 'Undersøgelser angående spedalskh. årsager,' Christ., 1874; Broch, 'Le royaume de Norvège,' &c., Christ., 1876, 52, and App. 7; Eklund, 'Om spetelska,' Stockh., 1879. See also 'Beretninger om Sundhetstilstanden i Norge,' and 'Tabeller over de spedalske i Norge.'

at the same time that the decrease has been nearly uniform all over.

Comparative Table of Leprosy in Norway in 1856 and in 1870.

Department.	1856.		1870.	
	Lepers.	Per 10,000 inhabitants.	Lepers.	Per 10,000 inhabitants.
Finnmarken . . .	16	8·3	12	5·9
Tromsøe . . .	58	16·1	30	6·6
Nordland . . .	275	35·4	231	25·8
Nord. Trondjhem . .	197	26·9	170	20·6
Søndre „ . . .	193	20·0	169	15·5
Romsdal . . .	336	37·2	288	27·6
Nord. Bergenhus . .	926	113·6	591	68·1
Bergen (Town) . .	47	18·2	26	9·4
Søndre Bergenhus . .	519	50·2	349	30·8
Stavanger . . .	225	24·6	157	15·0
Other Departments .	55	0·7	27	0·3
In the whole Kingdom.	2,847	19·1	2,050	11·7

On the *Farøe* and the *Shetland Islands*, as already remarked, leprosy has entirely disappeared since the beginning of the century. In *Iceland*,¹ on the other hand, it still persists, although there too it is considerably less than it used to be.

Table of Lepers in Iceland, 1768—1869.

Year.	Inhabitants.	Lepers.	Proportion.
1768	38,000	280	73 in 10,000
1838	55,000	128	23·3 „
1848	?	66	—
1869	70,000	110	15·7 in 10,000

¹ Older authorities: Petersen, 'Den saakaldte islandske Skörbug,' Sorøe, 1769; Thorstensen, 'Bibl. for Læger,' 1830, ii, 91. Recent authorities: Schleisner, 'Island undersøgt fra et lægevidensk. synspunkt.,' Kjöbenh., 1849, 23; Hjalte-
telin, 'Sundhedskoll. Aarsberetn. for 1855,' 25, 1856, 424, 1859, 435, 1866, 443, and in 'Dobell's Reports,' 1870, 283; Finsen, 'Jagttagelser ungaende sygdomsforholdene i Island,' Kjöbenh., 1874, 53.

The considerable decrease of cases between 1838 and 1848 is explained by the fact that many of them were swept off by the severe epidemic of measles in 1847. Between 1854 and 1859, the number rose again materially, but in 1869, as the above table shows, the proportion of lepers in 10,000 inhabitants was only 23·3, as against 67·3 from 1768 to 1838. The disease has always been most common on the south coast.

The *Western Hemisphere*, as we have already seen, was invaded by leprosy first in the seventeenth century, in connexion, it has been thought, with the importation of negroes. *North America*, however, has remained free from it, if we except the somewhat wide diffusion of the disease in *Mexico*, the occurrence of it among Chinese immigrants in *California*,¹ and two smaller foci, the one in *Louisiana*² and the other in *New Brunswick*. (The statement that leprosy occurs in *Greenland* rests upon errors of diagnosis, as more recent inquiry has shown.³) In *Louisiana* the disease has been endemic for a long time; in *New Brunswick* it is strictly confined to a few French settlements in the counties of Gloucester and Northumberland, between Chaleur Bay and the Miramachi, being properly endemic only in the village of Tracadie. The disease did not show itself there until 1815, and it is highly probable that it was imported by French *emi-grés* ("Acadians") from the coast of Normandy, and has been propagated among them by inheritance. The British Government had their attention first drawn to its occurrence in 1844, and they caused a leper-house to be founded on Sheldrake Island, into which thirty-two patients (out of a population of 4000 in the infected districts) were admitted during the five years down to 1849. The leper-house was afterwards transferred to Tracadie, and sixty-four new cases were admitted down to 1863, at which date there were twenty-one lepers in it, the rest having died.⁴ Later information⁵ tells us nothing more than that

¹ Piffard, 'New York Med. Record,' 1881, March, 305.

² Schmidt, 'New York Archives of Med.,' 1881, Dec.

³ Lange, 'Bemaerkn. om Grönlands sygdomsforhold.,' Kjöbenhavn., 1864, 25.

⁴ See Skene, 'Lond. Med. Gaz.,' 1844, June, 353; Boyle, *ib.*, Aug., 609; Alexander, 'L'Acadie,' Lond., 1849, ii, 226, 'Report on Leprosy,' viii, 1-6, 203-207.

⁵ Welch, 'Lancet,' 1874, Dec., 795; Adams, *ib.*, 852.

the disease still continues, its extent not being mentioned.

In *Mexico* leprosy occurs in general diffusion, mostly indeed among the native Indians, both on the coast and at the more elevated if not even the very highest points.¹ From *Central America* we have only a few facts about the disease; in *Nicaragua* it is said to be almost unknown,² and there is no mention of it in the accounts of sickness from *Guatemala*, *Honduras*, and the *Mosquito Shore*;³ only in *Costa Rica* is it spoken of as occurring often,—in the valleys of Cartago and San José at elevations of 3000 to 5000 feet, the number of lepers being estimated at from 50 to 100.⁴

More uniformly diffused, and in some places more frequent, is leprosy in the *West Indies*, particularly in *Cuba*,⁵ *Jamaica*⁶ (where there were in 1861 some 800 lepers in a population of 440,000, and of these 41 in Kingston with 27,000 inhabitants), *St. Bartholomew*,⁷ *St. Kitts*⁸ (47 lepers in a population of 24,000 in 1861), *Neris*,⁹ *Antigua*¹⁰ (22 persons in a leper-house, the population being 36,400), *Guadeloupe*,¹¹ *St. Vincent*,¹² *Barbadoes*,¹³ *Trinidad*¹⁴ (50 inmates of the leper-house in 1861, population 83,000), and the *Bahamas*.¹⁵ On the

¹ Blacquiére, 'Journ. des conaiss. med.,' 1838, Nov.; Simpson (quoting Cheyne), 'Edinb. Med. and Surg. Journ.,' 1842, April, 410; Newton, 'Med. Topogr. of the City of Mexico,' New York, 1848; Luvio é Alvaredo, 'Opusculo sobre el mal de San Lazaro,' Mexico, 1852; Jourdanet, 'Le Mexique, &c.,' Par., 1864, 413; Heinemann, in 'Virchow's Arch.,' 1867, xxxix, 607.

² Bernhard, 'Deutsche Klin.,' 1854, Nr. 8.

³ Young ('Narrative of a Residence on the Mosquito Shore,' Lond., 1847, p. 26) speaks of "leprous" spots which had been seen in natives of the Mosquito Shore; perhaps the reference is here to the skin affection known as the "Pintas" (*vide infra*), which is endemic among the Mexican Indians and has often been confused with leprosy.

⁴ Schwalbe, 'Arch. der Heilkde,' 1865, Heft. 6.

⁵ 'Notes on Cuba,' Bost., 1844.

⁶ 'Report on Leprosy,' ix, 9; Fiddes, 'Edinb. Med. Journ.,' 1857, June; Report in 'Philad. Med. and Surg. Reporter,' 1868, Jan., 21.

⁷ Laurén, 'Svensk. Läk. Sällsk.,' Hdl. ii, 177; Goës, 'Hygiea,' 1868, xxx, 460.

⁸ 'Report,' 16.

⁹ Ib.

¹⁰ Ib., 19.

¹¹ Ruzf, 'Bull. de l'Acad. de méd.,' 1859, xxiv, 1051.

¹² Ib., and 'Report,' 24.

¹³ Ruzf, l. c., 'Report,' 26.

¹⁴ 'Report,' 38; 'Report of the Medical Superintendent [Espinete] of the Leper Asylum (of Trinidad) for the year 1874.'

¹⁵ 'Report,' 7; Sweeting, 'Med. Times and Gaz.,' 1860, Sept., 208.

other hand, the disease is but rarely seen on *Porto Rico*,¹ the *Virgin Islands*,² *Dominica*,³ *Martinique*,⁴ *Sta. Lucia*,⁵ *Grenada*,⁶ and *Tobago*;⁷ rarely also on the *Bermudas*.⁸

I am unable, in the absence of medical information, to say whether leprosy is still as prevalent as it was described to be (in a report⁹ dated 1823) in *Colombia*—in the districts of *Cartagena*, *Socorro*, *Pamplona*, *Bogota*, *Tunja*, and *Canasara*. For *Venezuela* also I have no recent information. There are, however, trustworthy accounts¹⁰ of its endemic occurrence in *Ecuador* (less on the coast or in the upper basin of the *Marañon*, than in the elevated regions), and at various parts of *Guiana*, which agree with the reports on the disease in those countries during the last century.

For *Cayenne*¹¹ and *Surinam*¹² there are no definite data as to the number of cases. In *British Guiana*¹³ in 1864 there were counted (exclusive of 131 patients in the leper-house at the village of *Mahaica*, and of 60 patients under treatment in a private hospital), 338 lepers, who were distributed throughout the various districts of the colony as follows:

Berbice 136 lepers in 24,119 population, or 5·6 per 1000.

Demerara 85 „ 62,195 „ 1·3 „

Essequibo 53 „ 27,959 „ 1·9 „

Georgetown 64 „ 29,174 „ 2·2 „

Taking the total population as 148,000, the number of lepers (529) in *British Guiana* in that year gives a proportion of 3·6 per 1000.

The headquarters of leprosy in South America is *Brazil*.¹⁴ With the exception of the provinces of *Maranhão* and *Rio*

¹ Thomas de Cordoba, 'Mem. geogr. de la Isla de Puerto-Rico,' Sanmiltan, 1831.

² 'Report,' 15.

³ *Ib.*, 21.

⁴ *Rufz*, l. c.

⁵ Levacher, 'Guide méd. des Antilles,' Par., 1840, 263, 'Report,' 22.

⁶ *Ib.*, 34.

⁷ *Ib.*, 36.

⁸ *Ib.*, viii, 6.

⁹ Restropo, 'Memoria . . . al primero Congr. constit. de Columbia,' &c., *Bogota*, 1823.

¹⁰ Echeverria, 'Bull. de l'Acad. de méd.,' 1851, xvi, Nr. 17, 'Gaz. méd. de Paris,' 1851, 329 (from his experience in the leper-house of *Quito*); Duploux, 'Arch. de méd. nav.,' 1864, Oct., 283; Galt, 'Amer. Journ. of Med. Sc.,' 1872, Oct., 395; Gayraud et Daumec, 'Montpellier méd.,' 1878, Aug., 97, Dec., 491.

¹¹ Noyer, 'Revue méd.,' 1834, May, 235.

¹² Hasselaar, 'Beschrijv. der in de Kolon. Suriname voorkom. Elephantiasis en Lepra,' Amsterd., 1835, 22; v. Leent, 'Arch. de méd. nav.,' 1880, Nov., 405.

¹³ Hancock, 'Lond. Med. Gaz.,' 1837, Oct., 'Report on Leprosy,' x, 42-48, 213-15; Milroy, 'Med. Times and Gaz.,' 1873, May, 575; Hillis, 'Lancet,' 1879, Oct., 589.

¹⁴ See Sigaud, 'Du climat et des malad. du Brésil,' Par., 1844, 157, 160,

Grande, where the disease is comparatively rare, the whole country is afflicted with it to a frightful extent, more especially the inland provinces of Matto Grosso and Minas Geraes and the coast province of San Paulo. According to Tschudi there are villages on the frontier between Minas and San Paulo, in which almost every family is "morfetico." In his report for 1840 to the Provincial Estates of San Paulo, the president says:¹ "C'est un spectacle digne de compassion, sur la route de Rio de Janeiro à Saint-Paul, de reconstruire, échelonnés, tant de malheureux infectés de la lèpre. A chaque ville, on trouve une cabane aux environs qui sert de refuge à ces proscrits de la société."

In the leper-house at Bahia (besides which there are two large hospitals for lepers at Rio de Janeiro and Pernambuco), 1029 patients were admitted from 1787 to 1842. These figures do not give even an approximate measure of the frequency of the disease, inasmuch as it is diffused mostly among the poor, who endure their lot with indifference and do not seek a refuge in the leper-houses.

From the southern provinces of Brazil, this area of leprosy extends over *Paraguay* and the northern parts of the *Argentine Republic*, particularly throughout the provinces of Entre Rios and Salta, stretching across the continent as far as the eastern frontier of Bolivia.² The disease does not appear to occur at all on the west coast of South America; this fact is explicitly stated by the authorities for *Bolivia*, and there is no mention of leprosy in the numerous accounts of the state of health in *Chili* and *Peru*.

Sporadic cases.—To conclude this geographical and historical survey of leprosy, it remains to add that accounts are continually being published of the occurrence of isolated

382; Rendu, 'Étude topogr. et méd. sur le Brésil,' Par., 1848, 122, Ref. in 'Gaz. méd. de Paris,' 1848, 625; Dundas, 'Sketches of Brazil,' Lond., 1852, 359; Tschudi, 'Wien. med. Wochenschr.,' 1858, Nr. 31, 1866, Nr. 40; Plagge, 'Monatsbl. für med. Statist.,' 1857, Nr. 10, 71; Wucherer, in 'Virchow's Arch.,' 1861, xxii, 345.

¹ Quoted by Sigaud, l. c., 164.

² Brunel, 'Observ. topogr. et méd. faites dans le Rio-de-la-Plata,' Par., 1842, 46; Lallemand, in 'Virchow's Arch.,' 1861, xxii, 341; Mantegazza, 'Lettere sulla America meridionale,' Milano, 1860-63, i, 100, 141, ii, 208; Masterman, in 'Dobell's Reports,' 1870, 382; Coni, 'Contribucion al estudio de la lepra anestésica,' Buenos Ayres, 1878 (see Virchow-Hirsch's 'Jahresbericht,' 1878, i, 339).

cases in many countries, such as England, Germany, France, and Italy, where the endemic disease has been long extinct. The origin of these cases is not referred to endemic centres, but they are taken as having without doubt developed autochthonously in the midst of regions otherwise exempt from the disease. Many of these accounts appear certainly to be open to a good deal of suspicion, inasmuch as the diagnosis is anything but sure ; others, however, admit of no doubt as to the nature of the disease, and these are just the cases which have an especial interest for the etiological inquiry, being well adapted, if due attention be given to them, to preclude onesidedness in estimating the morbid causes.

§ 3. THE ETIOLOGY MUCH OBSCURED BY NARROW HYPOTHESES.

“The investigation of the causes of diseases,” says Sir James Simpson, in his classical ‘Antiquarian Notes on Leprosy,’¹ “has, probably more than any other department of medicine, been marked by belief without evidence, and assertion without facts. The history of the opinions which have at different times been so freely offered and adopted with regard to the production of leprosy, and the numerous explanations which have been proposed with respect to the causes of its almost epidemic prevalence in Europe in the middle ages, and its nearly complete suspension in the same region of the world at the present day, might easily, if time and space permitted, be made to form a chapter highly illustrative of the above general remark.” These words of Simpson describe the fortunes of the etiological doctrine of leprosy in all ages down to the present day, with that fitness and exhaustiveness of expression which an impartial testing of the facts always makes it possible to use ; and we may say without any reservation that superficiality or inaccuracy in observing facts, and onesidedness or bias in judging of them, have nowhere obtruded themselves so much to swell the doctrine of morbid causes with empty hypotheses as in the etiology of leprosy.

¹ ‘Edin. Med. and Surg. Journ.,’ 1842, April, p. 407.

§ 4. INFLUENCE OF CLIMATE.

In seeking to make good this assertion, we come first to a criticism of the views that have been held as to the *influence of climate* on the genesis of the disease. Either these views have had regard only to the prevalence of the disease in lower latitudes, laying especial stress on *tropical climate*; or, taking due account of the fact that leprosy occurs both in the tropics and in the extreme north (Norway and Iceland) they have found the material factor in the etiology to be *extremes of temperature and frequent and rapid transitions of temperature along with high degrees of atmospheric moisture*.¹ How little justice there is in this will appear from a glance at the present map of leprosy, which shows that it is indigenous in all latitudes, equally the tropical and polar, and the temperate (Ionian and Ægean Islands, Iberian Peninsula, River Plate States, Japan, the Cape, and New Zealand). But the history, also, of the disease in past centuries tells no less against that view, a history of far-reaching, if not quite universal, diffusion over Europe from the twelfth to the sixteenth century, and of the malady appearing and spreading, as well as gradually becoming extinct, altogether independently of climatic conditions or states of the weather.

As bearing on this point, and on the question of the etiological factors of leprosy in general, it is especially noteworthy that the disease is not found to be uniformly diffused over wide stretches of country at those points of the globe where it is now prevalent; but that its visitation falls upon single districts or even single villages within these limits, while neighbouring districts or villages, subject to the same, or approximately the same, conditions of climate, are either exempt altogether or only slightly touched by it. Lastly, there is the consideration that the greatest incidence of the disease within the tropics does not by any means correspond to the more or less pronounced tropical climate of the affected locality.

Out of 174 parishes in the district of Lafoès (Portugal), which has leprosy endemic in it, there are many quite free from the disease, the

¹ Brassac, 'Arch. de méd. nav.,' 1866, March, p. 189.

sickness being confined, on the whole, to the eastern parts of the valley; yet there is no difference, as d'Almeida expressly states, between the weather-conditions of the several parishes. Leprosy in Crete, as Smart informs us, has its head-quarters on the high land of the interior, badly situated as regards climate, while it is rare in the coast towns enjoying a mild climate; on the other hand, in Ceylon, it is most widely diffused in the southern coast districts, while the inhabitants of the interior, and particularly of the hills, are almost entirely exempt from it. On the east coast of Sweden, leprosy is extinct all but a small remnant; on the Norwegian west coast, however, an important centre of the disease still persists; while a comparison of the conditions of climate at the two points reveals no such material differences as would serve to explain the extinction of the endemic at the one and its continuance at the other. In the Bombay Presidency, says Carter, leprosy is severest at two points, which prove to be most widely different as regards climate—along the stretch of coast of the Southern Konkan (with a sick-rate of 1 in 430 inhabitants), and on the table-land of the Deccan (with a sick-rate of 1 in 550); and he adds:¹ “The disease is found under all climatic conditions, whether of soil, vegetation, temperature or rain-fall; its intensity has no ascertained relation to the sub-varieties of climate in Western India, and if it have to geographical conditions, this is only because they are connected with racial distribution.”

It should not be forgotten that unfavorable conditions of climate, high degrees of atmospheric moisture, frequent and extreme fluctuations of temperature, and the like, predispose the human organism to the specific sickness by their disturbing effects on the well-being; and herein, perhaps, there may lie *one* cause of the relative frequency of leprosy in the tropics. But in its *genesis* the disease is altogether independent of this factor in the etiology; its proper cause is to be sought rather in conditions which exert a *specific* effect, which are bound up with the locality or the manner of life, and very materially also with the racial characters of the inhabitants of the tropics, inasmuch as they make themselves felt there in a much greater degree than in higher latitudes.

Hansen² claims to find, in the fact that erythema nodosum, which some observers regard as a form of rheumatic sickness (?), is sometimes the precursor of leprosy, a reason for believing that the influence of chill is an important element in the production of the disease. The association herein implied I cannot regard as a perfectly good analogy; and

¹ ‘Med.-Chir. Trans.,’ l. c., p. 270.

² ‘Norsk. Mag.,’ l. c., p. 3.

Hansen himself admits that "it is hardly fair to assign the causation of the disease solely to influences of climate."

§ 5. QUESTION OF A SPECIAL RELATION TO THE SEA COAST.

The origin and endemic prevalence of leprosy cannot be brought into direct causal relation with definite *conditions of soil* any more than it can with climatic influences. The opinion held by many early observers, which was in currency until not so very long ago and was shared at one time by myself, that the disease mostly occurs on the sea coast and along the banks of rivers, and that we may make an inference therefrom as to the pathogenetic influence of a damp soil, can no longer be maintained, at least in its full extent, now that we have got more accurate information of the range of the disease within the modern period and at the present day, and that information, too, not only for the soil of Europe, but also for many other parts of the globe. It cannot, certainly, be denied that the greater part of the more considerable leprosy-areas still existing in Europe, do possess the character of soil above referred to; in Norway, in Spain and Portugal, and on the Ligurian and Adriatic littoral, we find leprosy confined practically to the coast; and the same is true for many centres of the malady in India, China, Ceylon, Egypt, Brazil, and the West Indies. But to set against such facts bearing out the rule, there are many more which do not accord with it; and the latter are so numerous that we come to doubt in the end what is rule and what is exception. Or rather—and this seems to me to be the correct standpoint from which to judge of the facts—we come to abandon the rule altogether, and admit that no definite factor of the pathogenesis can be found in states of the soil taken by themselves, that is to say, in anything therein that is physically discernible—the high or low elevation, the situation on the coast or in the interior, the dampness or dryness, the porosity or impermeability, and the like.

In speaking of the distribution of the disease in *Sicily*, Profeta¹ has the following:—"That which a considerable

¹ L. c., p. 297.

number of writers have taken to be a condition for the development of leprosy, namely, residence on the sea-coast, does not find confirmation in Sicily. In fact, while there are only 2 lepers to 9000 inhabitants of the coast, the proportion is about 5 among the population living at a greater or less distance from the sea." On *Madeira*, leprosy occurs at elevations from 2000 to 3000 feet (Heinecken). In *Abyssinia*, the disease is just as prevalent on the plains and in the hill-country (particularly the mountainous district of Samen), as on the coast (Pruner, Aubert-Roche, Blanc). In *Madagascar*, it is met with as commonly among the hills as on the flat country (Davidson). Of the distribution of the disease among the Hottentots in *Cape Colony*, we read:¹ "It is common at the Cape, principally among the Hottentots and half-castes The Hottentots usually reside away from the sea, in open valleys, high and dry, not liable to malaria." In *Syria*, leprosy is rare on the coast, and properly endemic only in the mountainous districts or on the plains (Pruner, Wortabet). In *India*, the head-quarters of the disease are the mountainous tract of Kumaon with an elevation of 5000 feet and upwards (Lewis and Cunningham); in the hill-country belonging to the Bombay Presidency, leprosy occurs at a height of 2000 feet, upon perfectly dry and scantily watered soil (Carter), and, under the same circumstances, on the plateau of Mysore (Van Sömeren). In *Sumatra*, the disease is found as an endemic mostly in elevated parts of the interior, notably in the beautiful park-like country of Paya-Combo.² In *Japan*, the area of leprosy extends from the coast far into the interior (Wernich). In *Mexico*, the table-land is afflicted no less than the coast (Jourdanet). In *Costa Rica*, it occurs nowhere but in the mountain valleys of Cartago and San José, at elevations of from 3000 to 5000 feet (Schwalbe). Of leprosy in *Barbadoes*, Carrington says:³ "It shows itself in all parts of the island, in towns, rural districts, on the sea coast and inland, in low damp situations and on dry hills." In *Colombia* and *Ecuador*, it is endemic only on elevated parts of

¹ Ebdén, in 'Report on Leprosy,' pp. 49-50.

² 'Arch. de méd. nav.,' 1867, Oct., p. 248.

³ 'Report on Leprosy,' p. 30.

the interior, while the coast regions, particularly those of Ecuador, are exempt. In *Brazil*, it is found to a frightful extent in the interior (especially in the province of Minas), and much more widely diffused than on the coast (Tschudi).

I think that, in adducing these facts, to which many more of a like significance from the most various parts of the world might have been added, I have said enough to justify the opinion that I have already expressed. The evidence should at the same time serve to show how entirely groundless is the opinion, which has been already controverted by Carter, Espinet (for Trinidad), and Durand-Fardel (for China),¹ that leprosy can be brought into some causal connexion with *malarious influences*.

§ 6. INFLUENCE OF FISH AS THE STAPLE FOOD.

The opinion that the true cause of the disease is to be looked for in the immoderate *use of fish as food*, or of *salt fish*, or of *fish that has become putrid or has undergone some morbid change*, is obviously connected with the foregoing assumption of the sea coasts being the chief seats of endemic leprosy. This view, which is foreshadowed in Galen,² and in the Arabian and other physicians of the middle ages (Gordon), as well as in the medical chroniclers of later centuries,³ has found its staunchest representative most recently in Hutchinson.⁴

"All localities," says that author, "which either are now, or ever were, noted as the homes of leprosy, have this in common, that they are either on the sea-shore, or on the banks of marine estuaries. The most probable conjecture is that it is caused by some peculiar diet common to marine localities; that it is due to fish eaten in some peculiar state, may be plausibly suspected. The fact that it is met with in such widely distant parts, renders it improbable that it is due to any particular variety of fish. . . . The sum of our conjectures, then, appears to amount to this: that leprosy is far too specific and peculiar in its symptoms to allow of our supposing it due to the influence of general poverty; that the cases in which Europeans are attacked, all

¹ 'Gaz. méd. de Paris,' 1877, No. 33, p. 493.

² *Vide supra*, p. 5.

³ Thus, Heberden ('Lond. Med. Transact.,' 1768, i, 23) states that the use of salted or putrid fish was the cause of the prevalence of leprosy in Madeira.

⁴ Not having the original before me, I quote the passage from Liveing, 'Elephantiasis Græcorum or True Leprosy,' Lond., 1873, p. 81.

indicate the power of endemic influences; that, of endemic influences, food is the one which has the most of probability as to its being the true cause; and, lastly, as the disease is only met with near the sea, we may plausibly guess that it is in some way connected with the fish diet."

In a subsequent paper,¹ in which he adduces observations from Norway, Russia, and India in support of this view, he remarks that leprosy has obviously spread along the shores of the Pacific in company with the Chinese immigration: "wherever they go the disease appears, and the explanation seems to be that, wherever they go, they carry their habits with them, and especially that they establish themselves everywhere as cooks; they are skilful cooks, and they can make use of many things which no one else would look at; decomposing fish and potted fish are amongst the delicacies in which they deal."

Numerous other writers have quite recently adopted the same opinion, on the ground of observations made in Spain and Portugal,² Sweden,³ Norway, Iceland,⁴ the Steppes of Southern Russia,⁵ Corfu, Crete, Ceylon,⁶ Japan,⁷ Abyssinia,⁸ New Brunswick,⁹ Guiana,¹⁰ and the West Indies.¹¹ They support their opinion by various arguments: either that, in the localities in question, fish, and more particularly salt or putrid fish, is almost the only food of the people; or that the disease has died out when the practice of subsisting on fish had been given up (as on the Bohuslän coast of Sweden); or, finally, that the fishes themselves had been subject to a specific malady, resembling leprosy, so that the eating of fish so tainted had set up the disease in man. It will be seen at once how one-sided and untenable this theory is, when we consider that leprosy is endemic in many places where there can be no question of a fish diet at all, and still less of the exclusive use of that kind of food; that the disease in many regions is limited as an endemic to particular localities, the population of villages in the immediate neighbourhood being quite exempt; that leprosy has disappeared

¹ 'Med. Press and Circular,' 1880, August 11.

² Grasset, Baptiste, Peacock.

³ Huss.

⁴ Mackenzie, Schleisner.

⁵ Oldekop.

⁶ 'Report on Leprosy.'

⁷ Schmid.

⁸ Blanc.

⁹ Skene.

¹⁰ Bergeron, Milroy, in 'Med. Times and Gaz.,' 1873, May, 575.

¹¹ 'Report on Leprosy.'

from many places not merely as an endemic but altogether, although no material change has taken place in the kind of sustenance of the inhabitants which would account for its disappearance ; and that the alleged communicable leprous disease of fishes has turned out to be a perfectly harmless parasite upon them, being found in many localities where no leprosy occurs.

With reference to the opinion that the use of decomposed fish, or of the greasy flesh of sea birds, is the true cause of the leprosy that is prevalent on the Norwegian coast, Danielssen says :¹ "The coast population, who suffer mostly from the disease, are in the habit of eating the fish in its fresh state or well salted ; in preparing the seagulls, which are by no means a general article of food there, most of the fat is taken away with the skin. Many persons fall into leprosy there whose diet is only exceptionally fish or the flesh of sea-fowl, their every day food consisting rather of meat and cereals." As to the "leprosy of fishes," Messrs. Boeck and Danielssen remark :² "Careful inquiries have shown that these suspected 'tubercles' are formed of a vegetable parasite, which is found in some species of soles even at places where there is not a trace of leprosy ; the people are afraid to eat the fish disfigured in that manner, and throw them away. The parasite has not the very slightest connexion with leprosy." Of this particular etiological factor the opinion of Profeta, based on his observations in Sicily, is : "Residence on the coast, with a seafaring life, or the prosecution of fishing and the use of salted or putrid fish, are regarded by a good many writers as the conditions likely to generate leprosy ; but this opinion is not borne out by the facts as regards Sicily." By far the larger number of authorities in India are opposed to the notion that the disease stands in any definite relation to a fish diet.³ Thus, Richards points out that in Balasore (Orissa), leprosy is less common than in Burdwan, Bancoora, and other districts adjoining, although the use of fish as food is much more universal in the first-named than in the others ; Macnamara⁴ says that,

¹ 'Norsk. Mag.,' 1851, v, 147.

² 'Traité,' p. 31.

³ See 'Brit. Med. Journ.,' 1880, April, 527.

⁴ 'Virchow's Arch.,' 1861, xxii, 312.

of all the cases of leprosy which he saw in India, there was not one for which this causal element had to be taken into account. Startin¹ mentions that the inhabitants of Rawalpindi (Punjab), and of the Naga Hills (Assam), eat no fish, and yet suffer from leprosy ; whereas in Araccan, where that article of diet plays a prominent part, the disease is rare. In Rutnagherry, one of the chief seats of leprosy, it is much more prevalent, as Carter points out,² in the interior, where no fish is eaten, than on the coast. It is remarked by Kirkpatrick³ that, in Bangalore (Mysore), leprosy is anything but rare among the Brahmins, although they are strict vegetarians, their partaking of flesh or fish being visited by immediate expulsion from the caste. Observations to the same effect come from various parts of the leprosy regions of China (such as Hankow), where, according to Shearer's experience,⁴ the use of fish food has no significance whatsoever for the genesis of the disease, and where leprosy, as Reid⁵ tells us, is often met with among the poverty-stricken population of the interior who live mostly on rice, millet, or other vegetable food prepared with a little oil, and who eat flesh only now and then, and fish more rarely still. In Hankow, on the other hand, and other centres of traffic, where fish is an article of diet the whole year round, the disease is rare. "Fish eating," Reid adds, "is essentially in Central China the privilege of the well-to-do, while leprosy is usually found among the poor vegetable-feeders." The Japanese physicians also, as we learn from Wernich, have declared against any connexion between living on fish and the development of the disease. When Skene refers the occurrence of leprosy among the Acadians of New Brunswick to their kind of food, Welch makes the reply that the English and Indian inhabitants of that region have just the same kind of diet as the former, and yet remain free from the disease.

Moreover, it is not a fish diet only that has been blamed for causing leprosy, but also other articles of food, such as

¹ 'Lancet,' 1880, Oct., 692.

² *Ib.*, 1872, Aug., 199.

³ *Ib.*, 1880, Dec., 922.

⁴ 'Lancet,' 1871, Dec., p. 801.

⁵ *Ib.*, 1880, Nov., p. 878.

*decomposed rice*¹ or *maize*.² But, most of all, a specific importance for the development of the disease has been ascribed to the *immoderate use of pork*, particularly in Brazil, where Candido³ would explain its prevalence in the provinces of San Paulo and Minas by the fact that swine's flesh forms one of the chief articles of the people's food. On the other hand, we have the recent statement of Lucio and Alvaredo⁴ from Mexico: "There are many lepers who have never eaten pork, others who have partaken of it rarely, and still others who have lived on it to an excessive extent; but among all these the disease has occurred with equal intensity. We conclude from this that the use of that article of diet has no influence whatsoever either upon the production of the disease or upon the severity of its type."

§. 7. INFLUENCE OF INSANITARY CONDITIONS.

But although there is no reason, so far, to look for the real cause of leprosy in dietetic influences, still, the predisposition of individuals to take leprosy appears to be very considerably heightened by *deficient, unsuitable, or bad food*, either in itself or along with other *defects of well-being*, such as inadequate shelter from the weather, filth, and the like. There is almost complete unanimity on this point among observers at the most diverse points of the globe. The opinion is further confirmed by the fact that the disease has been at all times especially prevalent among the necessitous classes, and much more occasional within those strata of society which are well found in the needs and comforts of life. We cannot, therefore, summarily dismiss the conjecture that the extinction of the disease at many places, or the considerable remission in its amount, has been a result of progressive improvement in hygienic conditions in the course of years, particularly of improved cultivation of the soil, and of a greater acreage;

¹ In India (Brett, 'Essay on some of the principal Surgical Diseases of India,' Calcutta, 1840, p. 163).

² In Brazil (Sigaud, p. 382).

³ 'Revista med. flumin,' 1842, p. 501. Tschudi also mentions that this opinion is widely held in Brazil ('Wien. med. Woch.,' 1858, l. c.).

⁴ L. c., p. 29.

for the more abundant harvests, bringing a better kind of sustenance, and more of it, have been a gain which cannot be too highly estimated in this respect. But these are etiological factors which one must not over-value. On the one hand, cases of leprosy have been not so very rare, both of late and in past times, among individuals of the well-to-do class, removed from the harmful influences in question; and, on the other hand, the malady has disappeared from many localities which used to be much afflicted by it, although the most wretched hygienic conditions are found in them still; while, finally, it should not be forgotten that, within the regions where leprosy is or has been endemic, there are particular villages, or even considerable tracts of country, quite exempt from the disease, although these nowise differ, in the manner of life of their inhabitants, from the parts of the country that are subject to the leprous infliction.

It is impossible to decide how far we can trust the statements¹ that have come down to us from the Middle Ages, as to leprosy occurring in crowned heads, princes, prelates, and other personages—whether, in many of these cases, it is not rather a question of syphilis. But there can be no doubt that the classes of the people in better circumstances furnished no inconsiderable contingent of the leprous in former times, and that they continue to do so in those countries where there are still intense centres of leprosy, such as Sicily,² Madeira and the Canaries,³ the North-West Provinces of India,⁴ Tirhoot,⁵ Orissa,⁶ Malacca,⁷ and Madagascar.⁸ The very narrow limitation of many of the leprosy-centres has already been dwelt upon more than once in the sketch of the historical and geographical circumstances. Vidal,⁹ in his account, dating from last century, of leprosy in the vicinity of Martigues (Provence), points out that the disease was confined to a few villages and to perfectly definite groups of families; and precisely to the same effect are the statements of Valentin,¹⁰ on its distribution in Pigna, Castel Franco, and other places on the Ligurian coast. Referring to the small focus of leprosy in Comaccio, Verga¹¹ says that the disease no longer occurs even at a short distance from the town in the directions of

¹ See Simpson, l. c., 1842, Ap., p. 394; Boeck and Danielssen, l. c., p. 97.

² Profeta. ³ Bolle. ⁴ Planck, 'Report.' ⁵ Macnamara.

⁶ Richards. ⁷ Official papers. ⁸ Borchgrevink.

⁹ 'Hist. de la soc. de méd. de Paris,' 1779, i, Mém., 188.

¹⁰ 'Bull. de l'école de méd. de Paris,' 1808, 45; also Foderé, 'Journ. complém. du dict. des sc. méd.,' 1819, iv, 3.

¹¹ 'Sulla lebbra,' Milano, 1846.

Ravenna and Forli. Similar observations have been made in the district of Söndfjord (Amt Bergen),¹ in the affected parts of Greece,² in Crete,³ at various places in India,⁴ at Hankow,⁵ in Japan,⁶ and elsewhere; and it has not been possible to find a reason for this in any difference between the affected and the exempted villages as regards the habits of living.

§. 8. THE DOCTRINE OF A SPECIFIC INFECTIVE VIRUS.

All these facts are opposed, as we have said, to the opinion, adopted by various inquirers, that the sum total of hygienic defects, and these *in and by themselves*, furnish the proper cause of the disease; or that leprosy ranges itself alongside of scurvy and the other *constitutional anomalies of nutrition*.⁷ Rather do the facts go to prove that this etiological factor has the significance merely of a predisposing cause, even if it be a potent one; that, for the production of the disease, we must have a definite and specific noxious agent, a peculiar infective substance, which had been more or less widely diffused through Europe in former times, but is now active only at certain points in this part of the world, although it still makes itself felt over a great range of territory outside Europe. Many observers have arrived at this opinion by a process of exclusion, being convinced that the production of the disease is not to be explained as an effect of atmospheric, dietetic, or other unhygienic influences, and that an *ignotum quid* must accordingly be invoked.⁸ Others, again, go a step farther, and develop the doctrine of the "*virus of leprosy*," and of its *infectious character*. Thus, it was said by Schilling in the last century :⁹ "Neque tamen

¹ Bidentkap, l. c., 1858, xii, 466. ² Dekigalla, Ornstein. ³ Brunelli.

⁴ Carter, 'Med.-Chir. Transact.,' l. c. ⁵ Shearer. ⁶ Wernich.

⁷ See, for instance, Vinkhuijzen, p. 110; Hjort, l. c., 1872, ii, 115.

⁸ This is the view of Boeck and Danielssen, l. c., 407-9 (Norway); Welhaven, 'Svensk. Läk. Sällsk. Handl.' iii, 188 (Sweden); Dundas, 369 (Brazil); Kaempfer, 161 (Madeira); Bargigli, l. c. (Mytilene); Wachsmuth, l. c. (Livonia); Profeta (Sicily); and Davidson (Madagascar). "If poverty in diet, or personal wants, filth, and wretchedness in their deepest degrees, could generate the malady," says Simpson (l. c., April, 409), "there are certainly still numerous spots in Continental Europe, and even in our own land, where, unfortunately, all these elements of disease are in our own day in full and active operation, without any such specific result following; the alleged causes are present without the alleged effects."

⁹ 'Diss. de lepra,' Traj. ad Rhen., 1769.

negaverim, peculiarem esse materiam et quasi virus quoddam, sine quo vera lepra non producatur." In later times, the same opinion has been expressed by Holmsen, Lockmann, and Hansen. To the last mentioned observer¹ the priority belongs of successfully demonstrating that there are organic elements in the morbid products, which are "perhaps" (as he cautiously puts it) the proper virus of leprosy.

He found uniformly, in the cells of the leprous nodules, "small rod-like bodies" which had the perfect type of bacilli; and, in the older morbid products, brownish cells of a larger size, which contained zooglæa-masses and aggregates of bacilli. Examination of the blood yielded no results at first; but when the device was adopted of keeping the preparation a few days in a moist chamber, there were found jointed filaments, which he was constrained to regard as likewise specific formations, inasmuch as they could not be made out in preparations of blood treated the same way, taken from healthy persons or from persons with syphilis.

These observations have been subsequently confirmed by Carter,² although with many modifications in points of detail, and more recently by Neisser,³ Cornil,⁴ and Köbner.⁵

Neisser found the bacilli not only in the leprous products, but also in the skin, in the mucous membrane of the mouth, gums and larynx, in the liver, spleen, and lymphatic glands, and in the interstitial prolongations of the peripheral nerves of the cornea, of cartilage, and of the testis. He satisfied himself, also, of a formation of spores proceeding from the break-up of the bacilli; and of the growth of the latter into filaments. Alterations in the blood at large Hansen and Neisser could in no case discover; and the latter is accordingly obliged to conclude that the diffusion of the spores within the body takes place by way of the lymphatic system. In blood taken from leprous nodules Köbner found bacilli; but he is not sure that they may not have got into the preparation owing to pressure exerted on the tissue.

In Neisser's opinion, the results of his inquiries have conclusively proved that in leprosy we have to do with *a specific*

¹ 'Undersøgelse, &c.,' Christian., 1874, 75, and in 'Virchow's Arch.,' 1880, lxxix, 32.

² 'Transact. of the Pathol. Soc. of London,' 1876, xxxvii, 297.

³ 'Bresl. ärztl. Zeitschr.,' 1879, Nr. 20, 21, and in 'Virchow's Arch.,' 1881, lxxxiv, 514.

⁴ 'Bull. de l'Acad. de méd.,' 1881, Nr. 43, p. 1308; 'Union méd.,' 1881, Nr. 134, 178, 179; 'Annal. de Dermatologie,' 1881, ii, 653 (along with Suchard).

⁵ In 'Virchow's Arch.,' 1882, Bd. 88, 282.

kind of bacterium, which can be brought into causal relation with every manifestation of the disease ; along with relapsing fever and anthrax, he says, leprosy is entitled to a place as one of the best authenticated bacterial diseases.

§ 9. QUESTION OF CONTAGIOUSNESS.

If the infectious nature of leprosy is rendered highly probable—I will not say proved—by the facts above adduced, I am nevertheless unable to share the opinion of those who have drawn conclusions therefrom, and particularly from the parasitic character of the disease, as to *its contagiousness*, and have thereby sought to rehabilitate with the credit of the very latest times a notion that had been almost entirely abandoned and condemned. During the Middle Ages, there was hardly any doubt among physicians and the laity as to the contagiousness of leprosy ; and it was that conviction which really gave occasion to the founding of leper-houses and to the isolation of the leprous. We shall, perhaps, not err if we attribute this assumption of a leprous contagium to an error in diagnosis, to the confounding of it with syphilis, the nature of which latter remained quite unknown to the mediæval practitioners. Pointing to this, at least, is the fact that as early as the 16th century, when men had learned to distinguish the one disease from the other, doubts about the contagiousness of leprosy became current. The adherents of the doctrine that leprosy was catching became fewer and fewer as the disentanglement proceeded and unprejudiced observation gained ground ; and, in the end, the belief was held only as a tradition within ever-narrowing circles. “Belief without evidence,” as Simpson says,¹ “and assertion without facts,” preserved this dogma in existence ; or, as Brunelli very aptly says : “L’opinione era contagiosa, e non la malattia.” In more recent times, the number of those who believed in it had dwindled to a minimum ; only among the public did “opinione” still play a considerable part, finding among the profession only a very few who were so cautious as not to

¹ *I. c.*, p. 412.

deny the possibility of leprosy being communicable through contagion. Thus it happened that Landré's coming forward as a contagionist,¹ although it made a considerable stir, was without result, the more so that he did not rely upon observations of his own, but took as the text of his declamations the extremely unreliable accounts which his father had brought from Surinam. The doctrine of the contagiousness of leprosy has been lately revived by Hansen,² who supports his opinion, in the first instance, by a series of cases where persons, previously in good health, took leprosy after coming into communication with lepers (which, of course, proves nothing, as all the cases occurred within endemic foci of the disease). But he was doubtless materially strengthened in his conviction by the discovery of bacteria in the leprous products; and this surmise is borne out by the fact that Carter, who was still anti-contagionist in 1873, became converted to a belief in contagiousness in the following year, after he had witnessed Hansen's researches and become persuaded of the trustworthiness of that discovery. In like manner it is only an *a priori* proof of the conveyance of leprosy by contagion which Neisser adduces when he states, on the ground of the finding of bacteria and of the hypothesis therefrom deduced, that the malady is "contagious in its specific products," and, "contagious not only *directly*, but also *indirectly* by articles which serve to carry the bacilli of leprosy or their spores."

That the contagiousness of leprosy is not proved by these arguments, nor even made probable, goes without saying; while, against the notion, there are all those facts, positive and negative, from the history of the disease, about which the observer who rests on "his own experience" does not concern himself. In judging of the question before us, I would not lay any emphasis on the want of success attending the experiments of Danielssen,³ Hansen, and Köbner to inoculate the disease upon rabbits (of Köbner also upon rats, mice, guinea-pigs, pigeons, fishes, and monkeys), inasmuch as experiments of that kind can only have a correct signifi-

¹ 'De la contagion seule cause de la propagation de la lepre,' Par., 1869.

² L. c., p. 53.

³ 'Norsk. Mag. for Laegevidensk.,' 1871, i, 195.

cance when they are made upon animals whose susceptibility to the morbid poison we are otherwise assured of. I attach equally little value to the felonious experiment by which Bargigli attempted (also without success) to inoculate the ichorous matter from a leprous ulcer upon children six to eight years old,¹ inasmuch as it is not proved that the bacilli of leprosy do occur in the pus derived from leprous persons. Finally, the statement of Davidson, from Madagascar, that "leprosy is contagious only by inoculation and not by simple contact," amounts to nothing, as he has omitted to say on whom the inoculation is made, and how it is made.

Equally indecisive, in my opinion, are the data of those observers who refer the outbreak of the disease in the Western Hemisphere (Guiana, Brazil, &c.) to the negro importation. Not only is the information by no means guaranteed; but, even admitting it to be trustworthy, the fact might be explained in other ways than the spread of the disease through contagion; besides that all the more recent observers in those countries deny most emphatically that leprosy is contagious at all. Lastly, if negroes had introduced leprosy into South America and the West Indies, it would be difficult to understand why Central America, and most of all the United States, where there has been truly no lack of negro importation, have remained exempt from the disease.

More recently Macnamara has given a similar history of importation of the malady by the Chinese to Honolulu in 1848, which had been told to him by Hillebrand, a practitioner of that place; but this may be said to have turned out a fable, as Milroy, Boeck, and others had already hinted, inasmuch as Emerson, a later authority, refers the same affair to the year 1856, adding that more than 1000 of the natives of the island had subsequently become affected with leprosy.² The statement of a French missionary, Etienne Brosse, author of a tract "*La lépra est contagieuse*," which has been quoted by Piffard,³ reads almost comically;

¹ As this statement may sound somewhat incredible I give the author's own words: "Afin de fixer mon opinion [on the non-contagiousness of leprosy], j'entrepris d'inoculer la matière sanieuse d'un ulcère de lépreux sur des enfants de 6 à 8 ans de cette classe. Je ne pus obtenir que deux fois seulement (!) l'autorisation de pratiquer cette opération. Cependant ces deux opérations ayant été négatives, ma conviction était faite."

² 'Brit. Med. Journ.,' 1880, Sept., 401.

³ 'New York Med. Record,' 1881, March, 305.

the Chinese, he says, have brought leprosy to California, and there can be no doubt that they will give the disease to the inhabitants of the State: "There is not the shadow of a doubt but that it will pass from them to the other inhabitants of the country. Thus far it has been confined to the Chinese, but it certainly will not be restricted to them." We still await the fulfilment of this prophecy.

Lastly, even those cases where Europeans have acquired the disease after coming into close connexion with leprous patients, cannot be regarded as giving proof of the contagiousness of leprosy, for the reason that they might very well have fallen ill apart from this intercourse, it being always in such cases a matter of acquiring the disease within the endemic seats of leprosy. My conviction is that there is not a single fact which tells decisively and indisputably for the conveyance of the disease by contagion. On the other hand the facts are very materially against the notion, inasmuch as they entirely fail to accord with our whole experience of the mode of diffusion of the true contagious diseases. These facts I tabulate as follows:

(1) The extremely narrow limitation of leprosy in certain centres, often very small, while there is free communication between their inhabitants and the neighbouring population, and where the sanitary conditions are such as would especially favour the conveyance of the disease sooner or later. This applies to the malady as it is distributed over the steppes of Southern Russia, in the Caucasus, in the northern districts of Persia, and in New Brunswick, referring to which last Jeffries says:¹ "I do not know where non-contagiousness of leprosy is better proved than among those descendants of one French family that brought the disease to New Brunswick."

(2) The observation made not unfrequently of the malady being confined, at certain places with a mixed population, to particular races or nationalities, notwithstanding unrestricted social intercourse throughout the community. Thus, in the East Indian Archipelago, the Arabs enjoy a striking immunity, while the Malays, the Javanese, and the mixed races are heavily afflicted.² To show how much truth there is in the opinion stated above, that leprosy had been imported into Honolulu with the Chinese, we may take a fact of the

¹ 'Lancet,' 1875, March, p. 358.

² 'Arch. de méd. nav.,' 1867, Oct., p. 248.

same class, vouched for by several medical officials in Melbourne: viz. that there has not been a single case observed of leprosy communicated by the Chinese living there, notwithstanding that their intercourse with all other sections of the community is perfectly free.¹

Vinkhuijzen² quotes from an official report by Uhlig, who had seen much of leprosy in Surinam and afterwards in Batavia, the interesting fact that the Indian races do not suffer from the disease either at the one place or the other, although they mix with the negroes in the most intimate way. "Since I have been resident," says Uhlig in his report, "at the settlement of Batavia, near which there are 500 Indians living, I have seen some of the latter coming here every day, or leprous persons going to them; they eat and drink together out of the same vessels, they house together, they wear each other's clothes, and what is more remarkable, they smoke the same pipe or the same cigar, passing it from one mouth to another, and no one declining to take it. This relationship has subsisted as long as the settlement itself, that is to say, for upwards of forty years, and yet no Indian has been infected with leprosy."

(3) The fact that, in innumerable cases, the acquiring of leprosy by one member of a family has led to no other outbreaks of it in that family, and that, too, under insanitary conditions which must have been quite peculiarly favorable to its conveyance; while it has been so rare for both parents to take it, that the circumstance, when it has occurred, may be referred, not to conveyance of the morbid poison from one to the other, but to infection of both from a common source, or to an endemic influence in general operation.

I select the following from a very large number of observations bearing on this point. Wortabet³ mentions a case that came under his notice at Beyrout (Syria) in which a leprous woman lived for many years with her husband and children, doing all the household work (cooking, washing, and the like) so long as she was able, and yet the husband and children continued in good health. Brunelli⁴ adduces the fact that in Crete there were as many as 127 persons living among the lepers from year to year, without one of the number taking leprosy. Bidentkap⁵ informs us that, in 1857 in the Söndfjord district, leprosy existed in 148 families, in 132 of which only one parent was affected, both being leprous in the remaining 16. Manget,⁶ who is a believer in the contagiousness of leprosy, adduces a few cases which seem to him

¹ 'Australian Med. Journ.,' 1874, March.

³ 'Med. Times and Gaz.,' 1880, Oct., 445.

⁵ 'Norsk. Mag.,' 1858, xii, 477.

² L. c., S. 129.

⁴ 'Report on Leprosy,' 64.

⁶ 'Report on Leprosy,' 45.

to tell that way; but he is constrained to add: "I have known instances [in Guiana] where black women have cohabited for years with their husbands while labouring under confirmed and ulcerative leprosy, and have children by them, without manifesting the slightest trace of the disease." According to Planck's¹ information for the North-West Provinces of India, there were among 855 leprous marriages, 831 in which the leprosy was on one side only; among the remaining 24, with leprosy on both sides, there were only 11 in which the possibility of the disease having been conveyed could not be excluded; but the small probability of disease-communication in the latter cases will appear from the fact that, where leprous men had married two or three wives, not one of these had ever become infected. There are accounts to the same effect by Bolle for the Canaries, Benson² for New Brunswick, Saturnin³ for Trinidad, and Ebdén⁴ for the Cape.

(4) There has never been a case known, in which the physicians or nurses of a leper-house have caught the disease, although they mix with the patients without restraint, dress their sores, and sometimes even wound themselves in so doing.

Thus, Browne⁵ writes from Barbadoes: "None of those in attendance, during the last nine years, upon the inmates of the lazaretto have contracted the disease; and I, after receiving a wound from a knife moistened with the fluids of an inmate, have escaped, although the wound was followed by great constitutional irritation and loss of the finger." To the same effect is the statement of Saturnin⁶ for Trinidad: "Ulcers with ichorous discharge are dressed several times a day by the surgery man, who has been employed for twelve years at the leper asylum. The washerwoman, who has been there for sixteen years, and handles the clothes of the lepers, and the medical superintendent, delivering women in labour, amputating limbs, and performing other surgical operations, have escaped." Powell,⁷ writing from Mauritius, says: "I know two instances where medical men have wounded themselves in dissection [of lepers], but without any bad results;" and we have similar information from the leper-houses of Madras, Calcutta, and Cawnpore.⁸ Hende⁹ says that during the nine years when he had medical charge of the Nagpore prison, there was not a single case of conveyance of leprosy from the sick to the healthy, although the intercourse between them had been perfectly free. In the Java hospitals, where lepers are admitted along with other patients, there has never been a case of the communication of leprosy observed.¹⁰ Drs. Lucio and

¹ 'Report on Leprosy in the North-West Provinces,' 1876, and 'Brit. Med. Journ.,' 1880, April, 527.

² 'Report on Leprosy,' 1867, 4.

³ *Ib.*, 39.

⁴ *Ib.*, 50.

⁵ *Ib.*, 32.

⁶ *Ib.*, 39.

⁷ *Ib.*, 86.

⁸ *Ib.*, xlv, xlv.

⁹ *Ib.*

¹⁰ In 'Arch. de méd. nav.,' 1868, Sept., 165.

Morado make the following statement on the alleged contagiousness of leprosy, from their experiences in Mexican leper-houses :¹

“Si efectivamente fuera contagioso, inoculable, los empleados del hospital lo habrian contraido alguna vez, viviendo como vivere continuamente en los salas, durmiendo algunos dentro de estas, y estando la mayor parte del dia en contacto inmediato con los enfermos. Por otra parte, los lazarinos que han copulado con mugeres sanas, y al reves, jamas han trasmitido el mal per un contacto tan inmediato como este. En las autopsias, repetidas veces, los que las han practicado se han picado las manos, han seguido poniendo la herida que resulta del piquete en contacto con los liquidos del cadaver sin haber tenido jamas accidente alguno.”

This exemption of doctors, medical assistants, nurses, and others in leper-houses is confirmed by the reports of a number of other observers, —Köbner² for San Remo, Sweeting for the Bahamas, Lewis and Cunningham³ for Kumaon, and Pasqua for Chios.

(5) No cases have come to light hitherto, in which the disease has spread from the leper-houses to residents outside.

Bargigli mentions that, although the leprous poor in Mytilene leave the leper-houses and wander about the island as beggars, no conveyance of the disease by them has been made out. Boeck⁴ says that patients received into the leper-hospital of Bergen have had unrestricted intercourse with the town, but that, so far as he knows, not a single case has occurred of the disease having been communicated by them to the town's-people; he remarks at the same time that none of the attendants who have been employed in that hospital had become affected with leprosy.

(6) Lastly, among the numerous cases of leprosy in Europeans, who had acquired the disease in leprous districts (having been affected with it either at the time of their return to Europe or having developed it subsequently), not one has ever been the occasion of the disease spreading in the immediate neighbourhood.

To these arguments—unanswerable, as I think,—for the non-contagiousness of leprosy,⁵ I will add the result of the collective inquiry on the matter before us which the College

¹ L. c., 29.

² ‘Vierteljahrschr. für Dermatol.,’ 1876, iii, 12.

³ L. c., 71.

⁴ Quoted by Carter, ‘Report on Leprosy in Norway,’ 1874, 49.

⁵ Vinkhuijzen, who has come to the same conclusion, ends his elaborate inquiry on the point (p. 151) with these words: “Alles resumerende ontkennen wij dus geheel de besmettelijkheid der melaatschheid onder welke omstandigheden ook, zoo wij vermeenen, op degelijke gronden en ware feiten ons steuende.”

of Physicians of London addressed to a large number of practitioners in all parts of the world where leprosy is prevalent.

“The all but unanimous conviction of the most experienced observers in different parts of the world,” runs the report,¹ “is quite opposed to the belief that leprosy is contagious or communicable by proximity or contact with the diseased. The evidence derived from the experience of the attendants in leper-asylums is especially conclusive upon this point. The few instances that have been reported in a contrary sense either rest on imperfect observation, or they are recorded with so little attention to the necessary details as not to affect the above conclusion.”

Many other observers of recent date have arrived at the same opinion: among them are Rigler² (Turkey), Smart (Crete), Peacock (Portugal), Milroy³ and Uhlig⁴ (Guiana), Schmid and Wernich (Japan), Labonté (Mauritius), Profeta (Sicily), Echeverria (Quito), London (Jerusalem), Auboeuf (India), Durand-Fardel (China), and Hercouet (Tahiti).

§ 10. THE INFLUENCE OF HEREDITY.

There is only one kind of conveyance of leprosy which cannot be questioned, I mean that which takes place *by way of inheritance*. There is almost complete unanimity on this point among the observers of all times;⁵ there are merely certain shades of difference between them, as to how high this pathogenetic factor is to be rated for the spreading of the disease, and whether the disease is inherited as such, or whether it is only a predisposition thereto that we are concerned with, a morbid diathesis which inclines the

¹ ‘Report on Leprosy,’ 1867, lix.

² “As regards contagiousness,” he says (‘Zeitschr. der Wiener Aerzte,’ 1847, Jahrg. iii, Bd. ii, 275), “I can give my word that there is not a trace of it to be found here.”

³ ‘Med. Times and Gaz.,’ 1873, May, p. 575.

⁴ L. c. (quoted by Vinkhuijzen, p. 129).

⁵ The Arabians, as well as the mediæval physicians of the West, held very pronounced opinions about it.

individual to fall into the sickness, or makes him specifically susceptible to the morbid poison. The discussion of the latter question comes within the limits of our inquiry only in so far as it requires us to adduce evidence that leprosy may also develop altogether independently of this inherited disposition ; whereof more in the sequel. But in answering the first of the two questions, which it is clear we can only do by means of statistics, we are met by two serious difficulties. One of these is that many of the best series of observations have been collected in parts of the world where it is especially difficult to get reliable information as to the state of health in the family of the leper. The other difficulty is that by far the larger number of cases belong to the endemic seats of leprosy ; so that there are many cases with the doubt attaching peculiarly to them whether the persistence of the disease in the family is really due to heredity, or whether it should not rather to be set down to the fact that each case individually, and quite independently of the hereditary factor, had developed itself out of endemic influences. And in fact, that kind of scepticism as to the propagation of leprosy by hereditary conveyance has been expressed by some of the Norwegian physicians, especially by Hjort and Hansen, who were subsequently joined in 1874 by Carter—although he wrote, in 1873, “I am of opinion that heredity is the common cause of the complaint”¹—and more recently by Neisser, although he does not specially state the grounds of his belief. Hjort, directing his polemic chiefly against Lochman, who had declared hereditary or contagious conveyance to be the only mode of origin of the disease, remarks² that the data as to the diffusion of the disease by inheritance had at any rate been much overvalued, and that, if we are to attach as much importance to that factor as many were inclined to do, it would be impossible to explain the somewhat rapid extinction of the disease in the Faröe Islands, in Bohuslän (Sweden), and in other places. That criticism seems to me to be perfectly justified ; but it does not suffice to set aside altogether the large mass of positive facts which tell in favour of heredity,

¹ ‘Med.-Chir. Transact.,’ lvi, 276.

² ‘Norsk. Mag.,’ 1872, ii, 122.

and it only serves to prove how far we still are from a perfect insight into the pathogenesis of leprosy.

Hansen confined himself, at the outset,¹ to the statement that the heredity of leprosy, although there was much in its favour, could not always be proved, and that the question was to be treated as still an open one; but he afterwards² denied "hereditary" conveyance altogether:

"Only those diseases," he says in so many words, "which depend on defects of development, which are occasioned, therefore, or called forth by a non-specific cause, may be designated 'hereditary diseases.' Contrasting with them, are those diseases which have a specific morbid cause underlying them, and are usually characterised, be they contagious or non-contagious, by perfectly definite and typical disorders of the normal functions of the body. If the disease be a contagious one, it may communicate itself by its virus to the fœtus in the womb, but then it is not hereditary; if it be non-contagious, it cannot be conveyed to the fœtus in any way whatsoever."

It is clear that we have to do here first of all with a conflict of words. There is no "hereditary" syphilis, says Hansen, but that which is usually so called is an infection of the fœtus *in utero*—which comes to the same thing as a mental concept. But Dr. Hansen forgets that there is such a thing as a congenital morbid diathesis, in which there can be no question of infection, a disposition towards a definite kind of sickness, residing in the organisation of the individual and determined by inheritance³—such as scrofula or gout. His definition of "hereditary" is, therefore, too narrow, and his definition of "intra-uterine infection" too wide. I am inclined to think that if Dr. Hansen had admitted among his general pathological concepts the idea of "conveyance" of the disease instead of "contagion" of the same, he would very soon have found himself occupying the same standpoint as those whom he controverts.

The best ground on which to try this question is obviously afforded by the small, closely circumscribed, and therefore easily surveyed leprosy-spots, with a fixed population subject

¹ 'Nord. med. Ark.,' 1870, ii, 21.

² 'Undersögelser, &c.,' 1874, p. 20.

³ The heredity of leprosy is understood in this sense also by Bidentkap ('Norsk. Mag.,' 1860, xiv, 843, by Holmboe (ib., 1865, xix, 153), and by Boeck ('Nord. med. Ark.,' 1871, iii, 1).

to no changes, where the state of health in the several families may be learned with the least possible trouble and followed with certainty through a long series of generations. Areas of observation of that kind existed at the beginning of the century at various points on the coast of Provence, especially around Martigues,¹ in Comacchio, and in several of the coast districts of Sweden;² and we still meet with them on the Ligurian coast, in Sicily,³ in certain provinces of Spain and Portugal, in Southern Russia⁴ and the Caucasus,⁵ in Greece, among the Dutch population of the Cape,⁶ in the northern districts of Persia, on some of the islands of the East Indian Archipelago (especially Ternate)⁷ and in New Brunswick.⁸ For all these places we do in fact find, in the authorities quoted, classical proofs that the disease clings to particular families as a consequence of continuous inheritance from generation to generation, and that the extension and multiplication of these small centres of disease is due to intermarriages among members of leprous families and of families which had been hitherto healthy. An interesting contribution to the subject is supplied by the observations of Holmboe and of Boeck (ll. cc.) on the outbreak of leprosy among Norwegian emigrants to the United States, who had settled in localities (of Illinois and Minnesota) quite free from the endemic. In 18 cases of this kind in Minnesota, reported on by Boeck, the disease had begun to develop in 9 before they left Norway, and of those 9, 5 belonged to families in which there were other lepers (all in the collateral line) still living; the other 9 cases did not develop until some considerable time (two-and-a-half to fourteen years) after their removal to America, and in all of them but one there was proof forthcoming of leprosy among their relatives in Norway. The natural supposition is that we have here to do, either with an inherited disease lying latent for many years, or with a congenital morbid diathesis. But there are other similar accounts of leprosy spreading by means

¹ Vidal, Valentin, Fodéré, ll. cc.

² Profeta.

³ Huss.

⁴ Plachof.

⁵ Liebau.

⁶ Schwarz, 'Zeitschr. der Wien. Aerzte,' 1858, Nr. 40.

⁷ In 'Arch. de méd. nav.,' 1870, Mars, 176.

⁸ Skene, Welch.

of heredity, from all the great leprous regions; although, as we have seen, the opinions differ as to the extent to which this factor influences the production of the disease, the statistics collected to elucidate the point having an unequal value.

The heredity of leprosy was ascertained in Bergen by Boeck and Danielssen¹ for 185 out of 213 cases, by Holmsen in the sanitary district of Midt-Nordland for 55 out of 93 cases, by Bidentkap² in the Söndfjord district for 393 out of 538, by Brunelli in Crete for 76 out of 122, by Profeta in Sicily for some three-fourths of a total of 114, by Fiddes in Jamaica for 184 out of 213, by Hillis in British Guiana for 31 out of 188, by Schwalbe in Costa Rica for 12 out of 15, by Planck in the North-West Provinces for 20 per cent., by Richards in Orissa for 53·4 per cent., by Day in Cochin for some 40 per cent., and by Lewis and Cunningham in Kumaon for 35 per cent. of the sick. Among 17 leprous families who were living, according to the last-named observers,³ in the Kumaon Leper Hospital, and in 4 of which both parents were leprous, there were 68 children born, and of these 27 were already the subjects of leprosy at the time the report was written.

Thus the opinion of Simpson⁴ holds good to the present day: "*Few facts in the history of tubercular leprosy seem to be more universally admitted by all writers on the disease, both ancient and modern, than the transmission of the predisposition of it from parents to offspring.*"

§ 11. SPECIAL LIABILITY OF THE NEGRO AND OTHER RACES.

This predisposition to fall into leprosy, accruing from heredity, may be applied to explain, in part at least, the varying amount of the sickness met with *in different races and nationalities*, although doubtless the manner of life is also not without its influence. All observers agree that, in countries with a mixed population, such as the West Indies, British, French, and Dutch Guiana, the Bahamas, Zanzibar, the Mauritius, India, Ceylon, and the Argentine States, the disease is commonest among the negroes and those of mixed blood (mulattoes, sambos, mestizos), and rarest, if not absolutely rare, among the Europeans. At the Cape,

¹ 'Traité,' p. 334.

² 'Norsk. Mag.,' xiv, l. c.

³ L. c., p. 61.

⁴ L. c., p. 404.

similar differences are found between the Hottentots and the white residents,¹ and, in China, between the Chinese and the Europeans.² In Algiers it is mostly the Kabyles that suffer, the Arabs very much less;³ and, in Crete⁴ and the East Indies,⁵ the Arabs enjoy a certain immunity from leprosy. The same immunity is very notable among the Indian race in Surinam and in Java, in contrast to the severe incidence of the disease upon the negro population and the Malays.⁶ The differences in the frequency of the disease among the Jewish population in various parts of the world are very remarkable; while they come next to the negroes in their great liability to leprosy in the West Indies (Jamaica,⁷ St. Vincent)⁸ and in Surinam,⁹ they are but rarely subject to it in Syria (notably Damascus¹⁰ and Jerusalem),¹¹ in Chios¹² and Crete,¹³ at Bombay,¹⁴ Aden,¹⁵ and other places. That this is not to be explained altogether, as Hasselaar seems to say for Surinam, by unfavorable conditions of living, may be inferred from the statement of Bowerbank, who points to the enormous frequency of leprosy among the Jewish population of Jamaica, and adds: "The well-to-do and the poor Jews suffer equally."

§ 12. ACQUIRED BY EUROPEANS WHO HAD RESIDED IN LEPROUS LOCALITIES ABROAD.

However highly we may rate the element of heredity in the history of leprosy, there cannot be the slightest doubt that, in innumerable cases, the disease had developed, and still develops, *independently*, that is to say, *solely under the influence of the proper pathogenetic factor and quite apart from specific congenital predisposition*. Irrespective of those well-authenticated cases of the spontaneous development of

¹ Black, Kretzschmar, Ebdén, in 'Report on Leprosy,' 1867, xxx.

² Wong.

³ Guyon, Bertrand.

⁴ Brunelli.

⁵ In 'Arch. de méd. nav.,' 1868, Sept., 165.

⁶ Compare the account of Uhlig, quoted from Vinkhuijzen on p. 48.

⁷ Fiddes, l. c., Bowerbank, in 'Report,' xxix.

⁸ Sprott, ib.

⁹ Hasselaar, 22.

¹⁰ 'Report on Leprosy,' xxx.

¹¹ London, Langerhans.

¹² Pasqua.

¹³ Brunelli.

¹⁴ Carter, 'Report,' xxx; Waring.

¹⁵ Steinhauser, 'Report,' xxx.

leprosy among the indigenous inhabitants of its endemic seats, the most telling evidence is furnished by the cases of individuals who have acquired the disease after spending some time at a centre of leprosy, having been born of healthy parents who had either resided all their lives in parts of Europe that are free from leprosy, or had migrated from these to tropical countries where leprosy is prevalent. The literature is full of such cases;¹ and it furnishes us, moreover, with a few cases which give evidence of a spontaneous origin of the disease even in regions where leprosy as an endemic had been extinct for centuries.² These latter cases remain just as much a puzzle in their genesis as was the historic sickness itself in its extinction; we are here at the limits of our knowledge, and there is not a single *well-grounded* hypothesis to show us the way beyond.

¹ See Goguelin, 'Bull. de la Faculté de méd. de Paris,' 1810, 91; Larrey, 'Denkwürdigkeiten,' i, 170; Alibert, 'Journ. complén. du dict. des Sc. méd.,' 1818, i, 159; Lee, 'New England Journ. of Med.,' 1818, vii, 41; Bielt (from 'Gaz. méd. de Paris'), 'Lond. Med. Gaz.,' 1829, Sept., 481, 513; Thévenot, 'Traité,' 249; Kinnis, 'Edinb. Med. and Surg. Journ.,' 1844, Jan., 54; Boeck et Danielssen, 'Traité,' 339; Wilson, 'Lancet,' 1856; 'Brit. Med. Journ.,' 1866, Oct., 456, 1870, July 8; Deraze, 'De la lèpre des anciennes,' Strasb., 1866; Huet, 'Nederl. Tijdschr. voor Geneesk.,' 1868, i, 113; Lignerolles, 'Gaz. des hôpit.,' 1867, Nr. 128; Rees and Moxon, 'Guy's Hosp. Rep.,' 1868, xiii, 189, 1869, xiv, 248; Arnott, 'Transact. of the Pathol. Soc.,' 1869, xix, 35; Piffard, 'New York Med. Gaz.,' 1869, iv, 1; Soltmann, 'Zur lepra nervosa,' Diss. Berl., 1869; Squire, 'Med. Times and Gaz.,' 1870, March, 296, and 'Transact. of the Pathol. Soc.,' 1871, xxi, 403; Benson, 'Dubl. Journ. of Med. Sc.,' 1872, April, 290; Thoma, in 'Virchow's Arch.,' 1873, lvii, 455; Espinet, 'Rapport,' 1874; 'Lancet,' 1875, Feb., 199, March, 339; Southey, 'Med. Times and Gaz.,' 1875, March, 299; Startin, 'Lancet,' 1880, Oct., 692. Hercouet (l. c.) saw four cases of the disease in Tahiti in Europeans who had lived several years there.

² Berndt, in Kausch's 'Memorabilien,' iii, 210; Wilson, 'Lancet,' 1856; Busch, 'Annal. des Berl. Charité-Krankenh.,' 1858, viii Heft, 2, 9; Nourse, 'Med. Times and Gaz.,' 1865, Sept., 251; Steudener, 'Beitr. zur Pathol. der Lepra mutilans,' Erlangen, 1867; Gaskoin, 'Brit. Med. Journ.,' 1873, Dec., 655, and 'Med. Times and Gaz.,' 1878, Jan., 86, May, 475; Langhans, in 'Virchow's Arch.,' 1875, lxiv, 175; Donor, 'New York Med. Record,' 1875, Nov., 20; Vidal, 'Gaz. des hôpit.,' 1875, 691; Kochler, 'Berl. klin. Woch.,' 1877, Nr. 46; Breuer, 'Vierteljahrsschr. für Dermatol.,' 1880, vii, 529, &c.

§. 13 THE ANÆSTHETIC AND TUBERCULAR VARIETIES.

As regards the relative frequency of *the two forms of leprosy—the anæsthetic and the tubercular*—at the several affected points of the globe, we are unable to judge with certainty from the data before us whether there are any very great differences. The anæsthetic form is in general more frequent than the tubercular ; it is very often the form in which the disease begins, tubercles appearing in the subsequent course and so producing the mixed form, while the inverse order is less frequent. If we take the statistics from a few leper-houses, giving the patients suffering from one form or the other, that relation would appear to be the general rule.

CHAPTER II.

SYPHILIS.

§ 14. ANTIQUITY OF ALL THE FORMS OF VENEREAL DISEASE.

THE history of venereal diseases, particularly of syphilis, has so often been made the subject of exhaustive inquiries, and the historical data bearing more or less directly on the occurrence of these maladies in former centuries have been so carefully collected, sifted, and critically elucidated in the writings of various investigators,¹ that I think I may decline to assume once more the whole armour of historical controversy. I shall content myself, accordingly, with putting together, in the following *résumé*, the conclusions which the facts, as at present ascertained, warrant concerning the *history of the venereal diseases in ancient and mediæval times*, and concerning *the epidemic outbreak of syphilis at the end of the fifteenth century*, referring the reader to the works quoted below, and particularly to latest comprehensive and thorough handling of the subject by Häser.

(1) Undoubted evidence of diseases of the genitals due to lewd or impure sexual gratification comes down to us from

¹ The principal authorities are: Astruc, 'De morbis venereis libri vi,' Paris, 1736 (and other editions); Hensler, 'Geschichte der Lustseuche, die zu Ende des 15 Jahrhunderts ausbrach,' Bd. i (the only vol. published), Altona, 1783 (1794); Thiene, 'Sulla storia dei mali venerei,' Venez., 1823; Huber, 'Bemerkungen über die Geschichte der venerischen Krankheiten,' Stuttg., 1825; Rosenbaum, 'Geschichte der Lustseuche im Alterthume,' Halle, 1839; Simon, 'Kritische Geschichte des Ursprunges, der Pathologie und Behandlung der Syphilis u. s. w.,' Th. i, ii, Abth. i, Hamb., 1857, 58; Friedberg, 'Die Lehre von den venerischen Krankheiten in dem Alterthume und Mittelalter u. s. w.,' Berlin, 1865; Güntz, 'Beitr. zur Geschichte der Medicin: über Alter und Ursprung der Syphilis,' Leipz., 1868; Müller, 'Die venerischen Krankheiten im Alterthum u. s. w.,' Erlangen, 1873; Häser, 'Lehrbuch der Geschichte der Medicin,' 3 Aufl., Jena, 1876-82, iii, 213-325.

every period of the world's history, even from biblical and mythical times.¹

(2) It is more especially the *venereal catarrh of the urethra (clap)*, and its consequences, whose occurrence may be followed with certainty into the remotest periods of antiquity.²

(3) Minute descriptions of ulcerous conditions of the male or female sexual organs are found not only in the ancient and mediæval compendiums of medicine and in prescription-books, but also in the erotic and satyric poets of Greece and Rome, as well as in many chroniclers of the Middle Ages; and these sores, when we bear in mind the frequency with which they occurred and the indications sometimes given as to their origin, cannot be regarded as other than venereal affections, that is to say, as *chancres, or primary syphilitic ulcers*.³

(4) There can be equally little doubt that *constitutional syphilitic affections* also occurred in antiquity and in the Middle Ages; although, for reasons to be mentioned in the sequel, the evidence relating to them is not so obvious as it is for the venereal diseases previously mentioned. In the descriptions given by the Greek and Roman physicians, of excrescences on the sexual organs and about the anus, which they designate by such names as *θύμοι, φύματα, κονδυλώματα, tubercula, pustulæ, or fici*, it is not difficult to recognise, among other morbid forms, the *flattened or syphilitic condylomatu*; and in the writings of the Middle Ages, whether medical treatises or lay chronicles, there is no lack of passages which afford us perfectly unambiguous evidence of the occurrence of *constitutional syphilitic disease*.

There are several things to explain the extreme scantiness of the knowledge of the venereal diseases in general which the ancient and mediæval practitioners of medicine possessed. Throughout the whole of antiquity, and even at the time when loose living had reached its highest development, a certain inversion of the moral sense restrained the public from confiding to medical practitioners those cases of disease which affected the parts of the body that they called *αἰδοῖα* or *pudenda*; to discover and expose these to the eyes of

¹ Häser, l. c., 218, 226.

² Ib., 219, 227.

³ Ib., 220, 228.

another person was regarded as disgraceful. The feeling was one that prevailed much more among the female sex than among men, and it is well known that the same feeling was the real hindrance to a knowledge of gynæcological and obstetrical subjects. It appears, moreover, that this aversion to a consultation with a medical practitioner in diseases of the sexual organs was not only a feeling that existed on the side of the compromised patients ; the doctors themselves, both in ancient and mediæval times, avoided them, preferring not to be occupied with so “ungenteel” maladies.

Another circumstance, connected with the former, which stood in the way of a correct understanding of the diseases in question, was the defective state of the etiological inquiry ; only the most oblique glance at the source and origin of these affections was tolerated—for obvious reasons. It seems to me to be quite certain that the Greek and Roman practitioners of medicine, as well as the contemporary public, knew of the connexion between sexual promiscuousness and the outbreak of these diseases, for there are many indications of this in the writings of the erotic and satyric poets of antiquity. But here again the reserve about such “secret things” prevented further inquiry into the question ; and the solution of it was all the less practicable for another reason, namely, that the idea of a fixed contagion was not acquired until a later date. All these matters were regarded *sub rosa*, and there was an unwillingness to call them by their right names. It is not until the writings of the mediæval physicians and chroniclers, that the source of these lesions of the sexual parts, together with the constitutional effects, begins to be quite plainly indicated. One of the most interesting references of that kind occurs in a manuscript of the thirteenth century by the Paris physician Gérard de Berry,¹ who observes, in his chapter “*de ulceribus et apostematibus virgæ* :” “*virga patitur a coitu cum mulieribus immundis de spermata corrupto vel ex humore venenoso in collo matricis recepto ; nam virga inficitur et aliquando alterat totum corpus.*”² It is obvious that this “*alteratio totius corporis*” means a constitutional malady associated

¹ Quoted by Littré, in ‘Janus,’ 1846, i, 593.

² See also Häser, l. c., p. 233.

with a preceding local infection of the *virga*. But unfortunately the author leaves us in uncertainty as to what the "alteratio" consisted in; and it is precisely that want of a correct and full understanding and description of the special phenomena arising out of constitutional diseases which has retarded the progress of inquiries into the ancient and mediæval history of constitutional maladies in general and of syphilis in particular.

The ancients were, at the outset, entirely without the notion of chronic constitutional sickness; their concepts of disease went altogether in the direction of a local pathology; and after the physicians of that period began to have some knowledge of one disease of that kind, when they made acquaintance with leprosy, they still set down (like the mediæval practitioners after them) all the varieties of local and general morbid process as an "outcome" (*Aussatz*), dwelling especially upon the process localised in the skin and mucous membranes. They had no single precise concept, no definite and comprehensive grasp of the group of symptoms distinctive of the disease; and, just as "plague" was their idea of severe epidemic sickness running an acute course, so they took leprosy as the representative of various chronic diseases. That syphilis played a leading part among the latter, we may infer from the opinion, widely diffused and passing unchallenged among mediæval practitioners, as to the origin of lepra "ex coitu cum foeda muliere." Thus Michael Scotus says:¹ "Si vero mulier fluxum patiat, et vir eam cognoscat, facile sibi virga vitiat, ut patet in adolescentulis, qui hoc ignorantes vitiantur quandoque virga, quandoque lepra." Gordon² says of the etiology of leprosy: "Et provenit etiam ex nimia confibulatione cum leprosis, et ex coitu cum leprosa et qui jacet cum muliere, cum qua jacuit leprosus;" and the same language is used by Gaddesden, Gilbertus Anglicus, Vitalis de Furno and others. This confounding of leprosy with syphilis has been the source, in my view, of the opinion which I have already mentioned (p. 7) as being held by contemporary writers, that "lepra" became widely diffused at the

¹ 'De procreatione hominis phisionomia,' cap. 6.

² 'Lilium medicinae,' Pars i, cap. 22, ed. Lugd., 1574, 95.

time of the Crusades and in consequence of them ; the same explanation applies to the doctrine held by several of those who witnessed the epidemic outbreak of syphilis towards the end of the fifteenth century—a doctrine rigidly maintained down to recent times—that the disease at that time began to develop out of leprosy, and that it is to be regarded as an “offshoot of lepra.” Thus Manardus¹ records the following opinion, which he thought a very credible one, of the outbreak of the malady in Spain and of its extension to Italy : “Coepisse hunc morbum (sc. Gallicum) per id tempus dicunt, quo Carolus, Francorum rex, expeditionem Italicam parabat : coepisse autem in Valentia Hispaniae Taraconensis insigni civitate a nobili quodam scorto, cujus noctem elephantiosus quidam ex equestri ordine miles, quinquaginta aureis emit : et cum ad mulieris concubitum frequens juvenus accurreret, intra paucos dies supra quadringentos infectos : e quorum numero nonnulli Carolum Italiam petentem sequenti, praeter alia quae adhuc vigent importata mala et hoc addiderunt.”

§ 15. THE GREAT EPIDEMIC OF SYPHILIS IN THE END OF THE FIFTEENTH CENTURY.

If these facts, then, justify us in concluding that the venereal diseases, and particularly syphilis, had not only occurred during antiquity and the Middle Ages, but were even by no means uncommon, the fact *that syphilis broke out in the form of a wide-spread and malignant epidemic towards the end of the fifteenth century* is still a very remarkable episode in its history. It is the strangeness of this outbreak that explains the enormous interest which the occurrence excited at the time, an interest which must have been all the greater that both the profession and the public found in it a form of sickness that was almost unknown to them. From that event we have to date the lively interest through which the attention of practitioners in all subsequent times down to the present has been enlisted, and elaborate inquiries into the causes of the outbreak have been set on foot.

¹ ‘Epistol. med.,’ lib. vii, Epist. ii, Basil, 1549, p. 137.

When and how this epidemic of syphilis began cannot be made out with certainty. This much only appears to be ascertained, that its first appearance was in the south-west of Europe, and that it spread thence with great rapidity and with a wide sweep over the Continent and the insular kingdoms.¹ So far as we may conclude from data before us, France was the starting point of the sickness; at least it is from that country that we have the first more or less trustworthy accounts of it for the period between 1488 and 1492.²

At the same time, or a little later (1493), it appeared in Spain, and in Italy in 1494.³ It spread over Germany and Switzerland in 1495 and 1496; in the Netherlands,⁴ as well as in Denmark and England,⁵ its outbreak was in 1496, and in Scotland⁶ in 1497. Our earliest information of the disease in Bohemia and Russia dates from 1499.

The extent of the disease in the various countries, or the number of persons attacked by it, is very variously stated.⁷ The duration of the epidemic can be estimated with equally little certainty; at some places the disease appears to have become extinct as early as the beginning of the sixteenth century, at others not until after the first twenty years of the same; and doubtless circumstances of locality and of social life were not without influence upon the duration of the sickness, as well as upon its intensity and severity. But it may at all events be inferred from the medical writings of the third and fourth decades of the sixteenth century, that syphilis had everywhere lost its epidemic character by that time, and was occurring in its milder forms; so that it finally

¹ See Häser, iii, 252.

² It is a noteworthy fact that the oldest designation of the disease, widely current in subsequent times and still met with in many countries outside Europe, is "*morbus gallicus*." For the various other names by which the malady has been known, see Häser, iii, 250. The term "*syphilis*" first occurs in the poem of Fracastori; it is derived, he tells us, from the name of a king's son, the shepherd Syphilus, who had blasphemed against the sun and was punished with the infliction of this disease.

³ The most recent information on the subject is given by Corradi, in the '*Giornale delle malatt. vener.*,' 1871, vi, 65, 145.

⁴ Fokker, '*Nederl. Tijdschr. voor Geneesk.*,' 1861, v, 451.

⁵ Mansa, '*Journ. for Med. og Chir.*,' 1833, Mar., p. 278.

⁶ Simpson, '*Transact. of the Epidemiol. Soc.*,' 1862, i, 144.

⁷ Sabellicus (quoted in the '*Aphrodisiacus*,' of Luisinus, Append. ed. Gruner, Jera, 1789, p. 116) puts it at one twentieth of the population.

assumed the type which it had probably borne in antiquity and the Middle Ages, the type in which it is found to occur at the present day.

§ 16. THE CHARACTERS OF SYPHILIS IN THE GREAT EPIDEMIC.

The descriptions left by contemporary writers¹ of *the type of syphilis at the time of its epidemic prevalence*, lead us to suppose that, while in very many cases ulcers on the genitals were the starting-point of the disease, yet the infection took place not unfrequently in other ways. We already find indications, also, of congenital syphilis in infants at birth.

Montesaurus² observes: "Plurimos enim vidimus, quibus in partibus pudendis nullum erat nocumentum." Cataneus³ says: "Quinta causa est longa mora et assidua dormitia cum infecta vel cum infecto sine coitu: vidimus enim quam pluries genetrices, filios suos tali modo infectos tractantes et eis ministrantes, post aliquod tempus infectionem hausisse. Hoc etiam modo vidimus pluries infantulos lactantes, tali modo infectos, plures nutrices infecisse." In like manner Torella⁴: "Si aliud membrum [in contrast to the genital organs] pustulum tangeret virulentum, aut sordidum, illud primo inficeretur, ut videtur in pueris lactantibus, in quibus prima affectio apparet in ore aut in facie et hoc accidit propter mammas infectas, aut faciem aut os matricis seu alicujus alterius . . . et saepius vidi infantem infectum hoc modo multas nutrices infecisse."

Among the local manifestations observed in the further course of the disease were condylomata, and scaly or tubercular exanthems, which often induced very considerable destructive ulceration; next to these, affections of the mucous membrane of the mouth and throat, which were also followed often by great loss of tissue extending even to the nose. Further, there is mention made of lesions of the skull and of the bones of the extremities, attended by unusually acute pains occurring especially in the night and robbing the patient of sleep; and, lastly, of affections of the eye, even leading to blindness. A few observers state, also, that the disease was sometimes diverted from the outer parts to the organs within; but it does not appear from the descriptions what was the nature of these visceral lesions. As to the

¹ See the detailed account in the first edition of this work, i, p. 337; also Häser, iii, 259.

² Quoted by Luisinus, p. 115 E.

³ Ib., p. 141 B.

⁴ Ib., 504 B.

duration and issue of the disease, it has to be said farther, that it lasted from several months to several years, and that one or more recurrences made it chronic. A perfect cure appears to have been rarely effected; in most cases the disease led to death, either suddenly in consequence of severe complications supervening (such as hæmorrhage), or under the symptoms of general cachexia due to gradual exhaustion.

§ 17. VENEREAL DISEASE IN FORMER TIMES IN OTHER PARTS OF THE WORLD.

The information relating to *the older history of the venereal diseases, and particularly of syphilis, in extra-European countries* is but slight. In the Ayur-Veda of Sûsruta there are several statements which make it at least very probable that venereal disease had occurred in *India* from the very earliest times.¹ Of the great antiquity of these diseases, particularly syphilis, in *China*, there can be hardly any doubt, according to the results of a thorough investigation into the subject made by Thin;² it is possible to trace the occurrence of the disease in that country as far back as the sixth century B.C. (time of Confucius), and we meet with a specific name for venereal sores in writings which date from the time of the Tang dynasty (618 to 906 A.D.). Dudgeon³ also traces the antiquity of syphilis in China back to the time of Confucius, and says that the disease at that time was prevalent in the Kingdom of Tei (now the province of Shan-Tung), whence it had spread southwards.⁴

From an interesting notice quite recently published by Scheube⁵ we learn that a detailed account of syphilis in its various forms is contained in a recently-discovered Japanese

¹ Friedberg gives the passages, taking them from Hessler's translation as revised by the well-known Sanscrit scholar Professor Weber. Wise's handling of the history of syphilis in India (in his 'Hindu System of Medicine,' p. 375) is quite untrustworthy, as Friedberg justly pronounces it to be.

² 'Edin. Med. Journ.,' 1868, July, p. 47.

³ 'Med. Times and Gaz.,' 1872, July, p. 56.

⁴ See also Morache, 'Annal. d'hyg.,' 1870, xxxiii, 25. The account by Dabry 'La médecine chez les Chinois,' Par., 1863), of the occurrence of syphilis in China in the time of the Hoang-Ti dynasty (*i.e.* 2600 B.C.), is, like the rest of his book, unworthy of any credit.

⁵ 'Virchow's Archiv,' xci (1883), p. 448.

medical work belonging to the ninth century, and that the authors of the treatise had been well acquainted with the link of connexion between these various local affections, recognising and correctly appreciating the constitutional character of the disease.

As to the earliest occurrence of syphilis in the *East Indian Archipelago*, I know only of the notice by Virchow, according to which the disease was introduced into the Moluccas and Philippines by the Portuguese in the beginning of the sixteenth century (1522?). I shall afterwards have to speak of certain places in this archipelago which have remained exempt from syphilis down to the most recent dates, as well as of the history of the disease in Oceania, Australia, and New Zealand, and in the western and southern divisions of the African continent.

One of the most interesting points which I have to mention in connexion with the question before us is the fact that syphilis did not occur in the Western Hemisphere until after the arrival of the Europeans, and as a consequence of importation of the disease. Dr. John Hunter,¹ speaking of the occurrence of syphilis in the West Indies, says: "Of the few things peculiar to this disease in the West Indies, it is perhaps the most singular that it should, at the present day, be much less frequent in a country supposed to have produced it, than in any part of Europe. This will not be considered as a proof that the venereal disease had its origin in the West Indies."

Clark² and Cordoba³ have subsequently expressed the same opinion, the latter remarking that the disease had been introduced into Porto Rico probably by the Spaniards. All the recent authorities in *Brazil* agree with Martius⁴ that those Indian tribes of the interior who have kept out of contact with European immigrants, enjoy an immunity from syphilis. The same observation has been made in *Paraguay*,⁵ on the pampas of *Peru*⁶ in the Sacramento Valley, and

¹ 'Observ. on the Diseases of the Army in Jamaica,' 2nd ed., Lond., 1796.

² 'Madras Quart. Med. Journ.,' i, 1839, Oct., p. 385.

³ 'Memor. geogr. . . . de la Isla de Puerto-Rico,' Sanmiltan, 1831.

⁴ 'Das Naturell, die Krank. . . . der Urbewohner Brasiliens,' München, p. 85.

⁵ Masterman, in 'Dobell's Reports,' 1870, 382.

⁶ Galt, 'Amer. Journ. of Med. Sc.,' 1874, Aug., 396.

among the Indian tribes in the interior of Northern California, whither the Spanish colonists had never penetrated.¹

Facts such as these are summed up by Jullien, in his inquiry into the geographical distribution of syphilis, in the following words:²—"Un fait indiscutable, c'est que la syphilis est aujourd'hui encore à peu près inconnue chez les peuplades, qui n'ont que peu de rapports avec les Européens, et que dans toute l'étendue des Amériques, c'est au développement de la vérole qu'il faut, toutes choses égales d'ailleurs, mesurer les progrès de la civilisation. Bien loin d'y avoir pris naissance, il est donc aujourd'hui prouvé que la syphilis est pour l'Amérique une maladie d'importation."

From the facts above stated, we may, in my opinion, conclude: *that the venereal diseases had occurred in Europe, and, so far as we know, also in various parts of Asia, from the earliest times; that syphilis overran a large part of Europe towards the end of the fifteenth century in epidemic-like diffusion, when it attracted the general attention of the profession for the first time and was first recognised by its peculiar features; that after the extinction of that epidemic, which lasted about thirty years, the disease fell again to its former level; that it was imported from Europe to other parts of the globe as a consequence of commerce between countries; but that even at the present day there are some places, remote from the general stream of traffic, which it has not reached (as we have already indicated and shall show at greater length in the sequel).*

But however remarkable that epidemic explosion of syphilis in the fifteenth century will always seem, it is by no means an isolated occurrence in the history of spreading sickness in general. Even in the after history of syphilis itself, we meet with a series of epidemic outbreaks, as we shall see presently, which, although they were confined to much narrower territories, present striking analogies with that event; and the inquiry into the circumstances under which these developed, and not unfrequently attained a vitality of many years, seems to offer the means of reaching more accurate conclusions as to the causes which were at work to produce that great pestilential outburst of the disease.

¹ Keeney, 'U. S. Army Statist. Report from 1855-60,' Washington, 1860, 241.

² 'Arch. de méd. nav.,' 1878, Aug., 150.

§ 18. PRESENT DISTRIBUTION OF SYPHILIS.

The *geographical area* of the venereal diseases at the present day, and particularly of *syphilis*,¹ extends over the greater part of the habitable globe. In the prevalence and intensity of the disease, however, we notice considerable differences at various points; differences which can be shown to depend in a definite way upon certain exterior conditions, above all upon the degree of culture and civilisation, and upon the rational foresight and concern of the population in things medical. Thus it happens that where these hygienic matters are either altogether or even in part in a backward state, syphilis is prevalent in its worst forms and has more or less the character of an endemic disease.

On *European soil*, the amount of syphilis is very nearly uniform in *Germany, Denmark, Austria, Switzerland, France, Northern and Central Italy, Greece, Spain and Portugal*. But, within these countries, there are local conditions, such as the luxurious life of great cities, the great development of industry, active commercial intercourse, and position on the seacoast, which make the differences in the amount of the disease not inconsiderable at the various points. In more extensive diffusion syphilis occurs in *Sweden and Norway, in Great Britain* (to judge by its frequency among the military), in *Southern Italy, in Sicily and in Turkey*.² It reaches its

¹ In what follows, I use the term "syphilis" in the sense of the unity doctrine: that is to say, I include therein both the so-called soft chancres, and the constitutional syphilitic disease. Apart from the fact that, in nearly all the data before us relating to the diffusion of syphilis in various parts of the world, we fail to find any separation between those two forms of disease (the distinction drawn being rather between the primary and the secondary syphilitic affections) I am myself of opinion that the dualist standpoint in the doctrine of syphilis is fundamentally wrong. There is undoubted evidence of the development of constitutional syphilis out of so-called soft chancres (see Köbner, 'Klin. Mittheil. aus der Dermatologie,' Erlang., 1864, p. 70, and the recent highly interesting paper of Rieger on cases from the clinic of Prof. v. Rinecker, in the 'Vierteljahrschr. für Dermatologie,' 1881, viii, 189). We have also the pathological curiosity which Professor Rollet has brought to light in his "chancre mixte," and the doctrine lately developed by Tarnowski ('Vierteljahrschr. für Dermatologie,' 1877, iv, 9) of the "pseudo-indurated chancre;" and these may be said to be bridges which the dualists have been constrained to build in order to rescue themselves from the dilemma that their doctrine has landed them in.

² Röser, 'Ueber einige Krank. des Orients,' Augsb., 1837, 67; Rigler, 'Die

greatest extent in many parts of *Russia*, such as the Baltic Provinces,¹ the Ukraine,² the Governments of Viatka,³ Samara,⁴ and Astrakhan,⁵ and the Kirghiz Steppe,⁶ as well as in *Finland*,⁷ and in *Roumania*. In *Montenegro*,⁸ on the other hand, syphilis is rarely met with; while the *Farøe Islands* and *Iceland* enjoy an almost perfect immunity.

An approximate measure of the amount of the disease in the several countries of Europe may be got from the statistical returns of syphilis among the soldiery.

Table of Syphilis in the various European Armies per 1000 men.

Country.	Period.	Admissions per 1000 men.
Great Britain ⁹	1860—69	236
Italy ¹⁰	1870—79	124
"	1864—65	120
"	1874—76	66
Bavaria ¹¹	1857—69	116
France ¹²	1862—69	106
"	¹³ 1872—73	85
Holland ¹²	—	105
Belgium ¹⁴	1858—60	100
Portugal ¹²	1861—67	96
Austria ¹²	1869	63
Prussia ¹²	1867	54

Türkei, &c., Wien, 1852, ii, 123; notice taken from the 'Gaz. méd. d'Orient' in 'Presse méd. Belge,' 1868, 155.

¹ Blum, 'Versuch einer Beschreibung der in Reval herrschenden Krankh.,' Marb., 1790, 148; Baer, 'Diss. de morbis inter Esthonas endemicis,' Dorp., 1814; Adelmann, 'Med. Ztg. Russl.,' 1844, Nr. 43; Attenhofer, 'Med. Topogr. von St. Petersburg,' Zürich, 1817, 246.

² Podolinski, 'Gaz. méd. de Paris,' 1881, Nr. 36, Feuille.

³ Jonin, 'Med. Ztg. Russl.,' 1849, Nr. 45.

⁴ Ucke, 'Das Klima und die Krankheiten der Stadt Samara,' Berl., 1863, 203.

⁵ Hirsch and Sommerbrodt, 'Mittheilungen über die Pest-Epidemie u. s. w.,' Berl., 1880, 9.

⁶ Maydell, 'Nonnulla topogr. med. Orenburg. spect.,' Dorpat, 1849.

⁷ Hjelt, 'Norsk. Mag.,' 1873, iii, 675.

⁸ Boulongne, 'Mém. de méd. milit.,' 1868, Dec., 486.

⁹ 'British Army Report for the Year 1879,' xxi, 11.

¹⁰ Sormani, 'Geogr. nosol. dell' Italia,' Roma, 1881, 226.

¹¹ Rothmund, 'Bayr. ärztl. Intelligzbl.,' 1872, Nr. 23.

¹² Laveran, 'Traité des maladies . . . des armées,' Paris, 1875, 446.

¹³ Granier, 'Lyon medical,' 1880, Nr. 18, 1.

¹⁴ Vleminkx, 'Gaz. méd. de Paris,' 1862, 445.

The wide prevalence of syphilis in Southern Italy and Sicily, in contrast to the Northern and Central Provinces of that peninsula,¹ had been remarked by earlier observers, and attention has lately been called to it by Celli² and Sormani.³ From the statistics given by the latter, relating to the prevalence of syphilis in the Italian army from 1874 to 1876, it follows that, while it averaged fifty-six per 1000 among the troops in the northern and central military divisions, it rose to eighty-eight in the southern division, and to seventy-eight in Sicily. The earlier accounts by Blaustein⁴ and by Barasch⁵ of the enormous diffusion of syphilis in Roumania, have lately been confirmed by the reports of Leconte⁶ and of Champouillon,⁷ the latter adding that a large part of the population, both old and young, urban and rural, are the subjects of the malady.

The Faröe Islands first received syphilis, according to Panum,⁸ by importation, in 1844; in the two years following, twenty cases came under observation, but in later reports nothing more is said of it. Into Iceland also, as Schleisner⁹ tells us, it has been imported twice (1756 and 1824). But the disease subsequently died out entirely; and although Schleisner gave the closest attention to the subject, he was not able to find a single case of primary or secondary syphilis on the island, notwithstanding the somewhat active sea-traffic maintained with it by means of Danish, French and Dutch vessels. These observations were afterwards entirely confirmed by Finsen.¹⁰

Among the regions of *Asia* most severely affected by syphilis, *India*, *China* and *Japan*,¹¹ having been the earliest

¹ Jansen, 'Briefe über Italien u. s. w.' From the Dutch, Düsseld, 1793, i, 297; Ziermann, 'Ueber die vorherrschenden Krankh. Siciliens,' Hannov., 184; Charlon, 'Gaz. méd. de Paris,' 1852, Nr. 5.

² 'Il Morgagni,' 1868, 800.

³ L. c., 104, 225.

⁴ 'Allgem. Ztg. für Chirurg.,' 1842, Nr. 49.

⁵ 'Wien. med. Woch.,' 1855, Nr. 49.

⁶ 'Considér. sur la pathol. des provinces du Bas-Danube,' Montp., 1869.

⁷ 'Mém. de méd. milit.,' 1868, xix, 177.

⁸ 'Bibl. for Laeger,' 1847, i, 316.

⁹ 'Island undersögt, &c.,' Kjöbenh., 1849, 2.

¹⁰ 'Jagttagelser, &c.,' Kjöbenh., 1874, 64.

¹¹ Cheval, 'Relation méd. d'une campagne au Japon,' Montp., 1868 32; New-

head-quarters of the disease, continue to hold the foremost place down to the present time. Next to them, there are very intense foci of syphilis in *Lower India*, especially *Cochin China* and *Laos*, where the disease is also said to have been prevalent in primæval times;¹ further, in *Kamtschatka* and *Siberia*, on many islands of the *East Indies*, on the coast districts and along the great caravan roads of *Arabia*² and *Persia*,³ on the littoral of *Syria*⁴ and on the table-land of *Armenia*.⁵

The authorities are unanimous in asserting the general diffusion and the frequency of syphilis in *India*.⁶ An approximate measure of the amount of this malady is furnished by the sick-rate of the British troops, of whom there were treated annually for syphilis (on the average of twenty years, 1860–79)⁷ 110 per 1000 in the Presidency of Bengal,

ton, 'Brit. Med. Journ.,' 1869, June, 521; Schmid, 'New York Med. Record,' 1869, Sept., 314; Potocnik, 'Arch. de méd. nav.,' 1875, Oct., 237; Manget, ib., 1877, May, 373; Godet, 'Étude sur l'hygiène au Japon,' Par., 1880, 46. In Japan, syphilis has been known from time immemorial by the name of "the fire of lewdness."

¹ Richaud, 'Arch. de méd. nav.,' 1864, April, 348; Beaufils, ib., 1882, April, 280; Thil, 'Remarques sur les princip. malad. à la Cochinchine,' Par., 1866, 34.

² Pruner, 'Krankh. des Orients,' 179; Palgrave, 'Narrative of a year's journey through Central and Eastern Arabia,' Lond., 1865.

³ Moore, 'Assoc. Med. Journ.,' 1856, Nov., 996.

⁴ Post, 'New York Med. Record,' 1869, June, 149; Barret, 'Arch. de méd. nav.,' 1878, August, 88. According to Robertson ('Edin. Med. and Surg. Journ.,' 1843, April, p. 247) it would not appear to have existed in the mountainous parts of the country until recent times when it was introduced by the troops of Ibrahim Pacha.

⁵ Wagner, 'Reise nach dem Arrarat,' Stuttg., 1848.

⁶ Macpherson ('Lond. Med. Gaz.,' 1841, June, 546) and Voigt ('Bibl. for Laeger,' 1834, April, 358), for Lower Bengal; Curran ('Dubl. Journ. of Med. Sc.,' 1871, Aug., 101), for the southern slope of the Himalaya; Leslie ('Transact. of the Calcutta Med. Soc.,' 1833, vi, 62) and McCosh ('India Journ. of Med. Sc.,' 1835, ii, 43), for Assam; Shortt ('Madras Quart. Journ. of Med. Sc.,' 1866, April, 262), for the District of Madras; Huillet ('Arch. de méd. nav.,' 1868, Févr., 87) and Auboef ('Contributions à l'étude de l'hyg. et des maladies dans l'Inde,' Par., 1882, 72), for Pondicherry; Shanks ('Madras Quart. Journ. of Med. Sc.,' 1839, i, i, 248, 20, 1841, iii, 13, 31), for Bellary, Secunderabad, and various other places in the Presidency of Madras; McGrigor (ib., 1842, iv, 159), for Bangalore; Day (ib., 1861, April, 326), for Cochin; McKay (ib., 1861, July, 29), for the Nilgherry Hills; Kinnis ('Edinb. Med. and Surg. Journ.,' 1851, April, 302), for Balgaum, Poona, and other places in the Presidency of Bombay; Gibson ('Transact. of the Bombay Med. Soc.,' iii, 68), for Gujerat.

⁷ Estimated according to the 'British Army Reports.'

117 in that of Madras, and 104 in that of Bombay. In *Ceylon*, the proportion is eighty-seven per 1000. Agreeing herewith are the reports of French¹ surgeons as to the frequent occurrence of the disease in *Cochin China*; in the hospital of Saigon, as Harmand² tells us, there are always some seventy or eighty venereal cases to be found among the 300 patients. Moreover, according to Thorel,³ syphilis is carried far into the interior of Laos by Chinese and Burmese caravans, and the only persons who escape it are the Anamese living in isolated settlements in the forests or in mountainous regions difficult of access. In *Kamtschatka*, syphilis is truly endemic,⁴ especially among the Koriaks and Tchoukchi; among the 300 persons forming the population of Petropaulovski, Maurin⁵ counted no fewer than thirty cases of inveterate syphilis. Referring to the diffusion of the disease in *China*, Morache⁶ says: "S'il était besoin encore de démontrer l'antiquité de la vérole, de repousser une fois de plus la doctrine un peu orgueilleuse de l'origine américaine, on pourrait en trouver des preuves dans son existence parmi les populations du nord de la Chine et surtout au milieu des tribus nomades de la Mongolie: la syphilis régné dans toute l'étendue de la Chine."

These statements are confirmed by the accounts of the prevalence of syphilis in Chinese ports,⁷ as well as by the information of Dudgeon⁸ for Peking and of Watson⁹ for Fung Thian (Southern Manchooria).

The range and frequency of syphilis in the *Islands of the Malay Archipelago*, as well as in *Australia* and in the *Islands*

¹ Richaud, *Thil*, ll. cc.; Girard la Barcerie, 'Considér. méd. sur la Cochinchine,' Montpell., 1868, 46; Gimelle, 'Union méd.,' 1869, Nr. 23; Sourrouille, 'Trois ans en Cochinchine,' Par., 1874, 17.

² 'Aperçu pathol. sur la Cochinchine,' Versaill., 1874, 47.

³ 'Notes médicales, &c.,' Par., 1870.

⁴ Bogorodsky, 'Med. Zeitung Russl.,' 1854, 10.

⁵ 'Arch. de méd. nav.,' 1877, août, 90.

⁶ 'Annal. d'hyg.,' 1870, xxxiii, 25.

⁷ Wilson, 'Med. Notes on China,' Lond., 1846, 26; Rochefort, 'Arch. de méd. nav.,' 1873, April, 281; Kerr, 'Edinb. Med. Journ.,' 1863, Aug., 189; Armand, 'Gaz. méd. de Paris,' 1861, 262, Feuille from Canton; Ref. in 'Arch. gén. de méd.,' 1866, Sept., 166, for Amoy; Cheval, l. c., 79, and Henderson, 'Edinb. Med. Journ.,' 1876, Nov., 405, for Shanghai; Rose, 'Pacific Med. Journ.,' 1862, Oct., for Foo-chow.

⁸ 'Med. Times and Gaz.,' l. c.

⁹ 'Edinb. Med. Journ.,' 1869, Nov., 442.

of the *Pacific*, can be shown to depend upon the more or less active traffic with other countries, and upon the efficiency of the police surveillance of prostitution in the Dutch and British possessions; so that a comparison of the amount of the sickness at the various points of this region brings out very considerable differences. In the *Malay Archipelago* it is most severe on the large and much frequented islands of Java, Sumatra, and the like; its widest diffusion is along the coasts,¹ although here again there has been an importation of it into the mountainous districts of the interior and a wide extension of its area.² Of the smaller island groups, those affected by the disease most are said to be the Riouw-Lingga Archipelago,³ and the Andaman Islands⁴ which have been used by the English (since the Sepoy Mutiny of 1858) as a convict settlement. In the Moluccas,⁵ where syphilis down to 1840 was no less widely prevalent than it was malignant, the state of matter is now materially better, thanks to the efforts of the Dutch Government to keep prostitution under control; this applies more particularly to Amboina,⁶ and less so to Ternate.⁷ The islands that suffer least are the small and isolated ones, almost out of the course of general traffic, such as the Nicobars,⁸ Banka,⁹ Billiton,¹⁰ the Banda¹¹ group and Timor.¹²

Syphilis is said to have been imported into *Polynesia*, particularly the Hawaiian or Sandwich Islands and Tahiti (Society Islands), by the crews of Captain Cook's ships and to have committed frightful ravages. As regards the Hawaiian group, it had been already stated by Lockwood¹³

¹ Heymann, 'Krankheiten in den Tropenländern,' Würzb., 1855, 187.

² v. Leent, 'Arch. de méd. nav.,' 1867, Oct., 246, 1874, Nov., 273.

³ Overbeek de Meijer, 'Nederl. Tijdschr. voor Geneesk.,' 1859, iii, 347; v. Leent, l. c., 1873, June, 412.

⁴ Brander, 'Edinb. Med. Journ.,' 1880, Nov., 394.

⁵ Lesson, 'Voyage méd. autour du monde,' Paris, 1829, 100.

⁶ v. Leent, l. c., 1869, Sept., 178.

⁷ Ib., 1870, March, 177.

⁸ Steen-Bille, 'Reise der Corvette Galatea um die Welt.' From the Danish, Leipz., 1852, i, 244.

⁹ v. Leent, l. c., 1873, Feb., 103.

¹⁰ Ib., 86.

¹¹ Ib., 1870, Jan., 14.

¹² Ib., 1870, Sept., 15.

¹³ 'Amer. Journ. of Med. Sc.,' 1846, Jan., 91.

and Gulick¹ that the disease has become a good deal rarer of late, being confined mostly to the shipping places; and although the most recent authorities² complain of its great frequency in Tahiti, yet we should take their statements, as Brunet³ does for the whole of Polynesia, as applying less to the natives than to the foreigners who come under hospital treatment. In other island-groups such as Fiji,⁴ Tonga,⁵ and Samoa,⁶ the disease is rare, or moderately diffused;⁷ and the same applies to *New Caledonia* where there had been no syphilis until the arrival of Europeans.⁸ It applies also to the *Australian Continent* and *Tasmania*; during the years 1821—1831 it happened to Scott⁹ to see only six cases of syphilis (imported) at Hobart Town, and it would appear from later information¹⁰ that the disease occurs there only to a moderate extent. In *New Zealand*, where syphilis is said to have been imported also by Cook's men (according to another account¹¹ not until the beginning of this century) and to have become widely diffused,¹² it now exists within moderate limits.¹³

The position of syphilis on the *continent of Africa and in the islands adjoining it* claims a peculiar share of interest in our inquiry. The disease has its chief distribution-area on the coast territories, to many of which it can be shown to have been imported from Europe; in the more inland districts

¹ 'New York Journ. of Med.,' 1855, March.

² Vauvray, 'Arch. de méd. nav.,' 1865, Dec., 527; Chassaniol et Guyot, *ib.*, 1878, Jan., 71; Hercouet, 'Étude sur les maladies des Européens aux îles Tahiti,' Par., 1880, 74.

³ 'La race Polynésienne, &c.,' Paris, 1876, 37.

⁴ Messer, 'Arch. de méd. nav.,' 1876, Nov., 321.

⁵ Wilkes, 'Narrative of a Voyage, &c.,' iii, 32.

⁶ *Ib.*

⁷ The statements made by several observers, that syphilis is common in these and other island-groups of Polynesia, have their origin in a confounding of the disease with chronic exanthems (the so-called Tonga-disease).

⁸ De Rochas, 'Topogr. méd. de la Nouvelle-Calédonie,' Par., 1860, 21; Charlopin, 'Notes rec. en Calédonie, &c.,' Montpell., 1868, 22.

⁹ 'Transact. of the Prov. Med. Soc.,' 1835, iii, App. xii.

¹⁰ Dempster, 'Calcutta Med. Transact.,' 1835, vii, 359; Hall, 'Transact. of the Epidemiol. Soc.,' 1865, ii, 84.

¹¹ 'Revue des Deux-Mondes,' 1879, 793.

¹² Power, 'Sketches in New Zealand,' Lond., 1849, 146; Thomson, 'Brit. and For. Med.-Chir. Rev.,' 1854, Oct.

¹³ Tuke, 'Edinb. Med. Journ.,' 1863, Sept., 227.

it is met with to a less extent, and in some places it is a modern or even quite recent intruder; while Central Africa, if not altogether free from it, is subject to syphilis only to a comparatively slight degree. These differences in the amount of the sickness are explained, as we shall see, not merely by the fact of no importation having taken place hitherto into certain parts of the continent, but also by other circumstances, which do not appear, however, to consist in the ethnological characters of the various nations.

One of the worst regions of syphilis in Africa is met with on the *East Coast*, and in the *East African Islands* of *Mauritius*¹ and *Réunion*,² but particularly in *Madagascar*, *Mozambique* and *Zanzibar*, where the number of the syphilitic is estimated at five-sixths of the whole native population,³ and from whence the disease has penetrated into the interior as far as the shores of Lake Tanganyika.⁴ Of the enormous diffusion of syphilis in Madagascar, not only on the coast but also in many mountainous localities,⁵ we have detailed information from Davidson⁶ and Borchgrevink.⁷

"Syphilis," says the latter, "har paa en forfaerdelig Maade gjennemtraengt det hele Folk. Fru Hofet og til den usleste Hytte findes Syphilis, og det ikke sparsomt, men rijeligt. Der vil neppe findes nogen Familie, hoor der ikke er sørgelige Spor af dens Virksomhed. Henved Halvparten af de under Behandling komne Syge ere i en eller anden Form Syfilispatienter."

In Madagascar, as well as on the adjoining islands of St. Marie and Mayotte, to which the disease is said to have been imported first in 1854,⁸ a remarkable phenomenon, vouched for by Borius⁹ and Dauvin,¹⁰ has to be noted: viz. that the disease among the indigenous inhabitants is almost exclusively confined to the Hovas, who are the dominant

¹ From the 'British Army Med. Reports,' 1859-66, it appears that the mean annual admissions for syphilis amounted to 122 cases per 1000 troops.

² Lesson, 'Voyage,' 144; Collas, 'Arch. de méd. nav.,' 1866, Nov., 405.

³ Lostalot-Bachoué, 'Zanzibar,' Par., 1876, 51.

⁴ Destrieux, 'Annal. de la Soc. de méd. de Gand., 1880, 78.

⁵ Vinson, 'Gaz. hebdomadaire de méd.,' 1866, Nr. 49, Feuill.

⁶ 'Edin. Med. Journ.,' 1863, March, 831.

⁷ 'Norsk Mag. for Laegvidensk.,' 1872, iii, Ser. ii, 242.

⁸ Daullé, 'Observ. méd. dans l'établissement franç. de Madagascar,' Par., 1857.

⁹ 'Arch. de méd. nav.,' 1870, Aug., 109.

¹⁰ 'Sur la syphilis à Réunion, &c.,' Montpellier, 1873, 9.

class of the population, and closely allied to the Malay race, while the Malagasy, who have the characters of the negro race in a marked degree, enjoy an almost complete immunity.

With reference to the occurrence of syphilis on the island of St. Marië, inhabited by the latter race, Borius observes: "Malgré la prostitution habituelle et générale des femmes, les accidents syphilitiques primitifs s'observent fort rarement, les autres affections vénériennes paraissent peu fréquentes." And Dauvin speaks still more decidedly as follows: "Les indigènes de Sainte-Marie malgré leurs rapports continnels avec les étrangers, tant Européens que créoles des îles voisines, Maurice et Réunion, où la syphilis est très répandue, ne présentent que très exceptionnellement les traces de l'infection Cette espèce d'immunité me frappa d'autant plus, qu'à une lieue à peine de Sainte-Marie, séparée par un chenal étroit et facilement navigable, la grande île malgache [Madagascar] est ravagée par la syphilis. Mais là encore la race noire est à peine atteinte, tandis que la caste de Hovas, d'origine malaise, semble lui offrir un théâtre tout préparé, où elle dérouté les diverses phases de son action."

Among the European settlers in the coast-regions of *Cape Colony*, syphilis is found to a considerable extent; and here again we observe the protective influence of certain circumstances of nationality against the development of the disease. Among the Hottentots syphilis is comparatively rare,¹ and it is still rarer with the Bechuana tribes farther towards the interior, notwithstanding that the disease has often been imported among them by the colonists;² while we learn from Livingstone that the negro races of unmixed blood, whose seats are in the central regions of *Southern Africa* towards the Kalahari Desert, are absolutely exempt from syphilis.

"A certain loathsome disease," he says,³ "which decimates the North-American Indians and threatens extirpation to the South Sea islanders, dies out in the interior of Africa without the aid of medicine. And the Bangwakatse, who brought it from the west coast, lost it when they came into their own land south-west of Kolobeng. It seems incapable of permanence in any form in persons of pure African blood anywhere in the centre of the country. In persons of mixed blood it is otherwise; and the virulence of the secondary symptoms seems to be in all the cases that came to my care in exact proportion to the greater or less amount of European blood in the patient. Among the Coronnas and Griquas of mixed breed it produces the same ravages as in Europe;

¹ Scherzer, 'Zietschr. der Wien. Aerzte,' 1858, 166; Schwarz, ib., 630.

² Fritsch, 'Arch. für Anat. und Physiol.,' 1867, 764.

³ 'Travels,' Lond., 1857, p. 128.

among half-blood Portuguese it is equally frightful in its inroads on the system; but in the pure Negro of the central parts it is quite incapable of permanence."

In *Abyssinia*, where the disease is said to have been imported by the Portuguese as early as the fifteenth century, it is now so universally prevalent through almost the whole of the country (but in the northern highlands and in the kingdom of Schoa, it is said, only since the beginning of the present century) that the number of those affected is estimated at nine-tenths of the population.¹ At the date of Rigler's information (1852), it would appear not to have penetrated into the country of the Gallas. There is but one opinion among the authorities as to the enormous diffusion of syphilis in *Egypt*² and *Nubia*;³ and the malady is prevalent to an equal extent, or in true endemic proportions, throughout the whole littoral of *Northern and Western Africa*—in *Tunis*, *Algiers*, *Morocco*,⁴ *Senegambia* and the *Guinea Coast*.

As to *Tunis*, Ferrini⁵ says: "Syphilis in this country is the most widely spread of all contagious maladies, the most serious and the most terrible;" and in like manner Rebatel and Tirant⁶ say of it there: "It reigns as sovereign in this country; those who do not have it are the rarest exceptions." *Algiers* forms one of the most frightful seats of syphilis, particularly, as Furnari⁷ tells us, since the French occupation;⁸ it penetrates as far as the Sahara,⁹ and reaches the maximum of intensity in the districts of Kabylia.¹⁰ Some

¹ Aubert-Roche, 'Annal d'hyg.,' xxx, 5; Rigler, 123; Courbon, 'Observ. topogr., &c.,' Paris, 1861, 35; Blanc, 'Med. Times and Gaz.,' 1868, Jan., 82, and 'Gaz. hebd.,' 1874, 350, Feuille.

² Pruner, 179; Griesinger, 'Arch. für phys. Heilkde,' 1853, xii, 517; Anelli, 'Annal. univ. di Med.,' 1871, Sept.; Vauvray, 'Arch. de méd. nav.,' 1873, Sept., 161; Nicoll, 'Annal. d'hyg.,' 1878, Sept., 210.

³ Veit, 'Württ. med. Correspdzbl.,' 1839, ix, 107.

⁴ Ref. in 'Med. Times and Gaz.,' 1877, July, 96; Dérugis, 'Voyage dans l'intérieur du Maroc,' Paris, 1878, 94.

⁵ 'Saggio, &c.,' Milano, 1860, 118.

⁶ 'Lyon médical,' 1874, Nr. 13, ii, 249.

⁷ 'Voyage méd. dans l'Afrique septentrionale,' Paris, 1845.

⁸ See Bertherand, 'Méd. et hyg. des Arabes,' Paris, 1855; Armand, 'Méd. et hyg. des pays chauds, &c.,' Paris, 1853, 415; Daga, 'Arch. gén. de méd.,' 1864, Sept.; Bertrand, 'Mém. de méd. milit.,' 1867, Mars, 199; Soyard, 'Considér. sur quelques-unes des affections . . . en Algérie,' Montp., 1868, 44.

⁹ Creissel, 'Mém. de med. milit.,' 1873, Juill., 337.

¹⁰ With reference to the disease known there under the name of "lèpre kabyle,"

evidence of the frequency of syphilis among the Europeans in *Senegambia* is furnished by the statistics of sickness in the French hospitals at St. Louis and Gorée; from the observations of twenty years (1853—1872) the mean annual admissions for venereal disease were 121 per 1000 men.¹ Among the natives, the disease is still more prevalent, being described by Hébert,² from observation of it in Dagana, as the “veritable scourge of the country.” According to Berger’s³ facts, there occurred from 1st January, 1862, to 31st July, 1865, in a battalion of Senegalese troops 812 strong, 585 cases of venereal disease, mostly syphilis, giving a mean annual sick-rate of 206 per 1000. As to the *Guinea Coast*, it is stated in an old account by Daniell⁴ (for the Benin and Biafra Coasts) that “syphilis is perhaps the most frequent and fatal of those maladies to which the male inhabitants are liable, and predominates more among them than among the females;” and more recent observers write to the same effect of the enormous amount of the disease on the Gold Coast⁵ and the Cameroon Coast.⁶ The statement of Balley is worthy of note, that in the Gaboon country syphilis is more rarely met with the farther one travels from the coast into the interior. Lastly, that syphilis is no stranger to the *Soudan* follows from the statements of Pruner as to the importation of the disease into Kordofan by Turkish troops, as well as from the accounts of its occurrence in Darfur,⁷

see Arnould, *ib.*, 1862, viii; Bazille, ‘Gaz. méd. de l’Algérie,’ 1868, 40; Challan, *ib.*, 117; Claudot, ‘Mém. de méd. milit.,’ 1877, May, 271.

¹ Béranger-Férand, ‘Traité des malad. des Européens au Sénégal,’ Paris, 1878, ii, 254.

² ‘Une année méd. à Dagana,’ Paris, 1880, 40. See also Borius, ‘Considér. méd. sur le poste de Dagana,’ Montpell., 1864; Chassaniol, ‘Arch. de méd. nav.,’ 1865, May, 518; Thaly, *ib.*, 1867, Sept., 185; Gauthier, ‘Des endémies au Sénégal,’ Paris, 1865, 18.

³ ‘Considér. hyg. sur le bataillon de Tirailleurs Sénégalais, 1862-65,’ Montpell., 1868, 59.

⁴ ‘Sketches of the med. topogr. of the Gulf of Guinea,’ Lond., 1849, 43, 96, 114, 138.

⁵ Clarke, ‘Transact. of the Epidemiol. Soc.,’ 1860, i, 112; Moriarty, ‘Med. Times and Gaz.,’ 1866, Dec., 663; Michel, ‘Notes méd. rec. à la Côte-d’or,’ Paris, 1873.

⁶ Griffon du Bellay, ‘Arch. de méd. nav.,’ 1864, Jan., 77; Abelin, ‘Étude méd. sur le Gabun,’ Paris, 1872, 31; Ballay, ‘L’Ogooué,’ Paris, 1880, 39.

⁷ Ebn-Omar-el-Junsi, ‘Voyage au Darfur,’ Paris, 1845.

and from the information of Quintius,¹ according to whom it has been imported quite recently from the West Coast into Segu-Sicorro (in 13° 32 N., 8° 26 W.). It is impossible, in the absence of further reliable data, to say how far syphilis extends through the Soudan generally.

In the *Western Hemisphere*, syphilis appeared first, as we have already remarked, in the sixteenth century, in consequence of importation from Europe; its diffusion there has followed the track of immigration and colonisation from east to west, so that, as Jullien aptly says,² the development of syphilis in America affords a measure, *cæteris paribus*, of the progress of civilisation in the several parts of that continent. To some regions, which had remained a long time remote from traffic, the disease has penetrated only in quite recent times; while still others continue exempt to the present day.

In the extreme northern parts of *North America*, we come first to two regions which, like Iceland, enjoy an almost absolute immunity. One of these is *Greenland*, where, in spite of prostitution flourishing to the utmost, and the busy traffic with Danish trading ships and British and American whalers, the disease is altogether unknown.³ The other is the island of *Miquelon* (in Fortune Bay, Newfoundland), where syphilis was introduced by the first immigrants, but took no permanent hold.⁴ In striking contrast hereto, is the great diffusion of the disease in the north-west of the continent, in *Alaska*,⁵ *British Columbia*⁶ and *Vancouver's Island*,⁷ where the native population are frightfully afflicted with the malady. Among the Eskimos inhabiting the Aleutian Islands, syphilis is said to have become considerably less common of late; on the other hand, in Vancouver's Island, to have become more general and very malignant in consequence of the complete neglect of sanitary police regulations. In *Canada*, where syphilis was imported from the

¹ 'Extrait d'un voyage dans le Soudan,' Paris, 1869, 39.

² 'Arch. de méd. nav.,' 1878, Août, p. 150.

³ Lange, 'Bemaerkn. om Gronlands Sygdomsforhold,' Kjöbenh., 1864, 30.

⁴ Gras, 'Quelques mots sur Miquelon,' Montpell., 1867, 39.

⁵ Blaschke, 'Topogr. med. portus Novi-Archangelensis,' Petropol., 1842, 66;
Ref. in 'Arch. de méd. nav.,' 1864, Dec., 475.

⁶ Simpson, 'Narrative of a Journey round the World.'

⁷ Maurin, 'Arch. de méd. nav.,' 1877, Aug., 93.

south at the beginning of last century, afterwards spreading destructively among the Indians,¹ and persisting down to the present in the form of severe endemics at various places ("Maladie de la Bay de St. Paul," and the "Ottawa disease"), it is now as prevalent as in the civilised states of Europe; and the same is true of the *United States*, where syphilis extends equally to the remnants of the Indian population wherever these have come into close communication with the European settlers.

Hunter,² who was in his youth carried off by a tribe of Indians living in the west, and who spent several years among them, says: "The venereal disease was entirely unknown among them [the Indian tribes] until they contracted it from the whites. . . . Those who go among the populous white settlements on the Missouri and Mississippi, where the disease prevails in its most inveterate forms among the traders and boatmen who navigate the river to New Orleans, frequently return to their families and tribes infected with it."

In California, it has become very common since the opening of the gold-fields, and it makes frightful havoc also among the Indians³; it was introduced by the Spaniards from Mexico, but it remained confined to the Indians of the south, the tribes living to the northward, whom the Mexicans did not reach, continuing down to the present day to enjoy an exemption from the disease.⁴ The state of matters is found to be the same among the Indian tribes occupying the Colorado plains, and the banks of the Missouri and the Red River; only those of them who have come in contact with Europeans or Mexicans suffer from syphilis.⁵ In Texas also the records show that, at the time when the territory was taken possession of by the United States, syphilis was found only among those Indian tribes who had trafficked with the Mexicans.⁶

Mexico, then, would appear to be the focus whence the

¹ Swediaur, 'Pract. Observ. on Venereal Complaints,' Edinb., 1788, 172; Stratton, 'Edinb. Med. and Surg. Journ.,' 1849, April, 276.

² 'Amer. Med. Recorder,' 1822, July, 412.

³ Praslow, 'Californien,' Götting., 1857, 56; Lantoin, 'Arch. de méd. nav.,' 1872, mars, 179.

⁴ Keeney, in 'U. S. Army Statist. Report,' 1855-60, Washington, 1860, 241.

⁵ Hoffmann, 'Philad. Med. and Surg. Reporter,' 1879, Feb., 160.

⁶ Hussion, in 'U. S. Army Statist. Report,' 1839-54, Philadelphia, 1856, 377; Swift, *ib.*, 385.

infection of the indigenous inhabitants of adjoining parts of North America had chiefly proceeded; and that country, in fact, takes a foremost place among the most intense centres of syphilis in the Western Hemisphere. The disease is prevalent there not only to an unusual extent, but also of a peculiar malignancy; whereof the surgeons of the French army had abundant opportunities of assuring themselves during the occupation of the country by French troops.¹ Syphilis is widely spread, also, in *Central America*—*Nicaragua*,² *Costa Rica*,³ *Guatemala*⁴ and *San Salvador*⁵—where it is again those Indian tribes that have kept out of contact with the immigrant population which remain free from it. It is widely spread in some of the *Antilles*, such as Hayti⁶ and Barbadoes;⁷ while in the larger number of them (Jamaica, St. Bartholomew,⁸ Martinique⁹ and others) it occurs only to a moderate extent. It is in *South America* that the disease reaches the maximum of diffusion and of intensity; and here too, the Indian population have kept free from it (some of them being still exempt) only for so long and in so far as they have had no intercourse with the immigrants from Europe.¹⁰

Of the frightful extent to which syphilis prevails in *Brazil* there are reports and complaints of the same tenour from all parts of that great territory.¹¹ Thus, from Bahia we learn:¹²

¹ Newton, 'Med. topogr. of the City of Mexico,' New York, 1848; Porter, 'Amer. Journ. of Med. Sc.,' 1853, Jan., 40; Jourdanet, 'Le Mexique, &c.,' Par., 1864, 412; Douillé, 'Montpellier médical,' 1872, août, 119; Heinemann, in 'Virchow's Arch.,' 1867, xxxix, 613, 1873, lviii, 177.

² Bernhard, 'Deutsche Klin.,' 1854, Nr. 11.

³ Schwalbe, 'Arch. für. klin. Med.,' 1875, xv, 342.

⁴ Bernoulli, 'Schweiz. Zeitschr. für Med.,' 1864, iii, 100.

⁵ Guzman, 'Essai d'une topogr. méd. de la république du Salvador,' Paris, 1869, 121.

⁶ Blacas, 'De la syphilis observée à St. Domingue,' Montpell., 1853.

⁷ Jackson, 'Boston Med. and Surg.,' 1867, July, 447.

⁸ Goës, 'Hygiea,' 1868, 460.

⁹ Rufz, 'Arch. de méd. nav.,' 1869, Nov., 351.

¹⁰ Compare the accounts by Varnhagen ('Hamb. Mag. für Heilkde,' 1822, iv, 367), and Martius ('Krank. der Urbewohner Brasiliens,' Münch., p. 85), for Brazil, by Masterman for Paraguay, by Pöppig for Chili, and by Galt for Peru.

¹¹ Rendu, 'Étude topogr. et méd. sur le Brésil,' Paris, 1848, 78; Sigaud, l. c., 117; see also Rey, 'Arch. de méd. nav.,' 1877, Jan., 28.

¹² Ref., ib., 1869, mars, 340.

“La syphilis est tellement commune dans toutes les familles, qu’on ne cherche nullement à la dissimuler, et on parle aussi volontiers du gallico dont est atteint un parent ou un ami, que s’il s’agissait d’un rhumatisme ou d’une attaque de goutte.”

In Pernambuco, according to Béringer,¹ there are in every 1000 deaths, 10 from syphilis. The accounts from *Paraguay* and the *Argentine States*² are similiar. “Patients with syphilis,” says Tschudi, “are found to a frightful extent all over the Argentine Confederation. Everywhere, even at the most remote posts, we find persons with the most horrible disfigurements of the face. In Cordova there are some dozens of them begging in the streets. Dr. Oster assured me that one would not be far out in taking every third person in Cordova for syphilitic.” But we meet with the disease in *Chili*,³ *Bolivia*,⁴ and *Peru*, as extensively as in the eastern states of South America. It sounds almost incredible to read in the account by Fournier,⁵ that of 912 deaths in the hospital of La Caridad at Valparaiso from May, 1871, to March, 1872, 52 were from syphilis (15 males and 37 females); and yet we shall be quite disposed to credit this statement on learning, as we do from Savatier,⁶ that of 972 patients admitted into that hospital in a year (1877-78), 485 were syphilitic. In *Peru*, where the disease is equally common,⁷ it was quite unknown before the Spanish conquest of the country (Tschudi); and even down to the present day it has not penetrated, according to Galt,⁸ into the pampas of Sacramento in the basin of the Ucayali.

¹ ‘Arch. de méd. nav.,’ 1879, mars, 222.

² Brunel, ‘Observ. topogr. et méd. faites dans le Rio de la Plata,’ Paris, 1842, 45; Tschudi, ‘Wien. med. Wochenschr.,’ 1858, Nr. 45; Mantegazza, ‘Lettere med. sulla America meridionale,’ Milano, 1860—63, i, 305, ii, 208; Masterman, in ‘Dobell’s Reports,’ 1870, 382.

³ Pöppig, in Clarus’ ‘Beitr. zur Heilkde.,’ 1834, i, 529; Lafargue, ‘Bull. de l’Acad. de méd.,’ 1851, xvii, 189; Accounts in ‘Arch. de méd. nav.,’ 1864, juli 22, août, 108; Boyd, ‘Edinb. Med. Journ.,’ 1876, Aug., 116.

⁴ Bach, ‘Zeitschr. für vergl. Erdkunde,’ iii, 543.

⁵ ‘Arch. de méd. nav.,’ 1874, Sept., 147.

⁶ *Ib.*, 1880, Jan., 14.

⁷ Tschudi, ‘Oester. med. Wochenschr.,’ 1846, 474; Lesson, ‘Voyage,’ 27; Accounts in ‘Arch. de méd. nav.,’ 1864, Sept., 181, 189, Oct., 274.

⁸ ‘Amer. Journ. of Med. Sc.,’ 1847, April, 400.

§ 19. HAS SPREAD OVER THE GLOBE THROUGH THE CONVEYANCE
OF A VIRUS.

A glance at this outline of the history and geography of syphilis shows us that the disease had been originally indigenous at a few points, and has gradually become diffused over almost the whole globe. From what points it issued, or what was *its native habitat*, we can form no more definite opinion than we can of the origin of the plague, the small-pox, typhus, and, indeed, of every other infective disease. This much, only, we may conclude with some probability from the data before us, that the disease existed in Europe and in Eastern Asia from the earliest times, that in the course of centuries it spread in ever-widening circles by means of the commerce between countries, that there were large territories, such as the whole of the New World, the centre of Africa, Australia and Oceania which it did not reach until comparatively modern times, or even quite recently, and that even at the present day there are regions exempt from syphilis where the population keeps itself free from communication with the infected country around. There can be no question that a *specific infective substance* underlies syphilis (which substance we may take to be an organic body from its power of reproduction), that the disease never develops autochthonously now, but occurs always in consequence of the conveyance of the morbid poison, and that this transmission may take place either by way of contagion, in the strict sense of the word, or by way of inheritance. Accordingly, with regard to the pathogenesis, the only questions that can arise are whether external influences such as climate, soil, and hygienic conditions, may perhaps determine the frequency and severity of type of the disease, or whether certain racial or national peculiarities may not sometimes afford a protection against it (relative or absolute) or at other times increase the predisposition of individuals to it.

There has naturally been no lack of inquiry into the nature of the specific *poison of syphilis*. Salisbury¹ was the first who professed to find it in a filamentous fungus (*Cryptos syphilitica*), which developed

¹ 'Amer. Journ. of Med. Sc.,' 1868, Jan. 17.

from spores; it took root in the connective tissue, extending its destructiveness to the tissues around, and, when the disease became constitutional, it could be detected also in the blood. Shortly afterwards Hallier¹ announced that he had discovered the parasite of syphilis in a micrococcus, which, when it was cultivated, grew into a fungus named by him *Coniothecium syphiliticum*. A few years later Losterfer² came forward with his discovery of "syphilis corpuscles" in the blood, but these were soon given up as illusory.³ Then came the announcement by Cutler⁴ that he had observed peculiar changes in cases of syphilis, the white corpuscles being "enlarged and distended by intercellular vegetations, the spores of which were copper-coloured," while the blood-serum contained "mycelial filaments" also copper-coloured. This discovery has obtained but scanty recognition at the hands of the commission appointed by the American Medical Association to inquire into it. The same year (1878) Klebs⁵ published the results of his microscopic study of syphilitic subjects, and of his experiments to infect animals; according to these there are rod-like moving bodies (*Helicomonades*) discoverable in syphilitic tissue, which, when cultivated, become spiral-shaped masses, and, if given to monkeys, produce the characteristics of syphilitic disease. This result was confirmed in essential points by Bermann;⁶ while Pisarewski⁷ found in the chancreous indurations a finely granular zoogloea-like mass packed into the spaces of the tissue, and consisting of small round particles, out of which he conjectures that the rods seen by Klebs (but not seen by himself) might develop.

§ 20. SEVERITY OF TYPE NOT SPECIALLY AFFECTED BY CLIMATE.

Whether the prevalence of syphilis is at all influenced by *circumstances of climate*, appears more than doubtful when we keep in mind the fact that it is almost equally common in all latitudes, and that many parts of the cold zone in both hemispheres are as much affected by it as temperate or sub-tropical or tropical regions; and although many countries of the temperate zone, especially in Europe, appear to

¹ 'Bayr. ärztl. Intelligenzbl.,' 1868, 233.

² 'Wien. med. Presse,' 1872, Nr. 4, and 'Med. Jahrb. der Wien. Aerzte,' 1872, 96.

³ See also Wedl, Neumann, and others, in 'Wien. allgem. med. Ztg.,' 1872, Nr. 7, 8, and Köbner, in 'Berl. klin. Wochenschr.,' 1872, 209.

⁴ 'Transact. of the Amer. Med. Assoc.,' 1878, xxix, 165.

⁵ 'Prager med. Wochenschr.,' 1878, Nr. 41.

⁶ 'The Fungus of Syphilis,' New York, 1880.

⁷ 'Centralblatt für Chirurgie,' 1880, Nr. 32.

be more favorably situated in this respect, the reason of that is to be sought, not in the climate but in the hygienic circumstances. I am equally unable to persuade myself that the climate has any influence on the severity, or on the greater or less malignity of the *type of syphilis*. There are, indeed, a series of data tending to show that syphilis has a milder type in warm or tropical climates; that it is not only slighter in its symptoms but also more quickly got over. These observations come from Italy,¹ Greece,² Turkey,³ the littoral of Syria (contrasting with the mountainous region),⁴ Persia,⁵ the coast of Abyssinia,⁶ the interior of Egypt,⁷ Tunis,⁸ the West Indies,⁹ the littoral of Mexico,¹⁰ and Peru.¹¹ But the data in question rest in part upon errors; and further, it is by no means made out that this comparatively mild course of the disease is actually dependent on conditions of climate; while, lastly, we have the well-established fact that many of the most intense centres of syphilis are met with actually in lower latitudes, and that in these it is not the Europeans only, as often asserted, but also the natives who suffer from the disease in its severest forms.

All the more recent authorities for Italy¹² state that syphilis runs a much more severe course in the southern provinces (and Sicily), than in the northern. Speaking of Portugal, Robertson¹³ and Wallace¹⁴ are agreed that the disease among the English troops was of no better type than in England. The malignancy of syphilis in Egypt and Nubia is vouched for by Griesinger,¹⁵ Veit, Brocchi, and others, contrasting with the account above mentioned, by Rebatel and Tirant, of the mild type of the disease in Tunis—an opinion derived, as they themselves admit, from only a few cases, and going directly against the experience of practitioners in Algiers. Ferrini¹⁶ gives it as not only the most widespread, but also the most dangerous and frightful of the diseases preva-

¹ Menis, 'Topogr. statist.-med. della provincia di Brescia,' 1837, i, 168.

² Röser, 'Krank. des Orients,' Augsb., 1837, 67; Olympios, 'Bayr. med. Correspondenzblatt,' 1840, Nr. 12.

³ Oppenheim, 'Volkskrankh. in der Türkei,' Hamb., 1833, 79.

⁴ Robertson, l. c.; Yates, 'Lond. Med. Gaz.,' 1844, Feb., 567.

⁵ Polak, 'Wochenbl. zur Zeitschr. der Wien. Aerzte,' 1856, Nr. 29.

⁶ Aubert-Roche, l. c.

⁷ Pruner, l. c.

⁸ Rebatel, l. c.

⁹ Ruzf, l. c., for Martinique.

¹⁰ Jourdanet, Heinemann, ll. cc.

¹¹ Tschudi, l. c.

¹² *Supra*, p. 71.

¹³ 'Lond. Med. Report,' 1818, June, 459.

¹⁴ 'Edinb. Med. and Surg. Journ.,' 1829, Jan., 79.

¹⁵ L. c., 517.

¹⁶ *Supra*, p. 78.

lent in that country. All authorities (Shanks, McGrigor, Kinnis, Aubœuf, and others) are agreed as to the malignant character of syphilis in India and the Malay Archipelago. Heymann says:¹ "Cases of syphilis are found in exceedingly large numbers all over the islands of the East Indian Archipelago. . . . While the extensive distribution of syphilitic disease cannot easily escape notice, its intensity attracts our attention in a still greater degree. Most of these affections are very obstinate and difficult to cure." Pop's account² is the same; and, in a subsequent paper,³ in which syphilis is spoken of as being prevalent in the East Indies "d'une manière terrible," he says: "The climate of the tropics does not appear to be very favorable to the treatment of constitutional syphilis." In like manner, Laure⁴ concludes that a tropical climate has a particularly injurious influence on the type of syphilis in Cochin China and China. Among the French military surgeons in Algiers, there is only one opinion as to the malignancy of syphilis there. Thaly says of the type of the disease in Upper Senegambia: "The sequelæ are very serious in this country;" and there are similar accounts from the Guinea Coast, from Brazil and other countries. Further, we may here once more recall the fact that, excepting in the Chinese ports where the Europeans suffer most from the severe forms of syphilis, the disease in all the tropical or sub-tropical countries mentioned above is just as malignant and as difficult to cure in natives as in foreigners, and, in some localities such as Algiers,⁵ is even of a more malignant type in the former.

§ 21. ALLEGED UNFAVORABLE INFLUENCE OF ALTITUDE.

It appears to me to be questionable whether the severity of syphilis on the table-lands of Armenia, Abyssinia and Mexico, contrasting with its alleged milder type on the plains, is due to the *elevation*, or, in other words, to the influence of altitude on the health of the inhabitants, as Jullien⁶ and Rey⁷ suppose; whether also, as Rey conjectures, we are here concerned with that "anémie des altitudes" which Jourdanet has observed on the Mexican plateau, and with a debility of the organism thereby caused. At all events I have no knowledge of any such "anæmia of altitude" on the plateaux of Armenia and Abyssinia.

¹ L. c., 187.

² 'Nederl. Tijdschr. voor Geneesk.,' 1859, iii, 25.

³ 'Arch. de méd. nav.,' 1867, Oct., 246.

⁴ 'Hist. méd. de la marine française, &c.,' Paris, 1864, 67, 143.

⁵ *Supra*, p. 78.

⁶ 'Arch. de méd. nav.,' 1878, août, 155.

⁷ 'Annal. de Dermatol.,' 1880, ii, Sér. i, 686.

§ 22. IMMUNITY FROM SYPHILIS IN CERTAIN COUNTRIES AND
AMONG CERTAIN RACES.

Highly remarkable, again, is the comparative immunity from syphilis which the inhabitants, or certain sections of the inhabitants, appear to enjoy in some parts of the world, notwithstanding the open intercourse with other countries and the abundant opportunities for infection. So far as we know at present, this peculiar state of things, which has been adverted to already in describing the geographical distribution of syphilis, may be observed in Iceland, in the island of Miquelon (Newfoundland), in Greenland, in the central regions of South Africa, and among the black population of Madagascar and the adjoining islands.

Syphilis can be shown to have been imported twice into Iceland, in 1756 and 1824, and to have infected the inhabitants to the extent of 22 cases in all. "When we consider," says Finsen,¹ "that Iceland is visited every year by hundreds of ships, partly Danish trading vessels, partly French and English whalers, which give rise to communication with the inhabitants in the most various ways, it must seem a piece of great good fortune that the natives have not been infected more often;" during the nine years which he (Finsen) practised on the island, he saw only 5 cases of syphilis, and these in strangers. As regards Miquelon, Gras says: "Syphilis came into the island with its first settlers but it has not taken root. I have not found a single trace of its having been actually developed; and, although I have found evidence of secondary and tertiary lesions among the older generation, I have never seen anything in infants which would justify me in believing that the malady had been transmitted by heredity." The following is Lange's literal statement for Greenland: "It is remarkable that there is absolutely no syphilis here; about the fact itself there is not the slightest doubt, and the strangeness of it will perhaps seem less when we remember that the same is true of Iceland. The circumstance is explicable simply and solely on the ground that Greenlanders, like Icelanders, have an immunity from syphilis;

¹ L. c.

for there is no lack of opportunities for infection. Greenland is visited every year by vessels from Denmark, at whose departure from the mother country there are certainly precautions taken to prevent the importation of contagious diseases ; but hardly a year passes, especially in the northern parts of the country, without several settlements having to provide quarters, for a longer or shorter period, to considerable numbers of men shipwrecked from British and American whalers ; and besides these there are other occasional visitors. Now, considering that prostitution is carried on, both on board ship and on shore, with an absence of restraint which baffles all belief, it certainly cannot be said that the Greenlanders have not had ample opportunities, during nearly a century and a half, of becoming infected with syphilis."

I have already quoted at length the observation of Livingstone on the immunity from syphilis which is enjoyed by the negroes of the southern regions of Central Africa. To this Fritsch adds the remark : " Syphilis is rare, and it occurs in Bechuana Land only in very scattered cases, mostly importations from Cape Colony ; still there are materials with which to controvert Livingstone's assertion that the disease does not hold with pure Ethiopian blood." I have already called attention to the accounts by Borius and Dauvin as to the relative immunity from syphilis of the Malagasys (negroes) in Madagascar, Mayotte and St. Marie, in contrast with the frequent and serious cases of it among the Hovas (of Malay race) who live in the same localities. Referring to the intercourse of the Malagasys of St. Marie with Madagascar, Dauvin says : " Les communications entre ces deux points sont journalières, la facilité des mœurs, le libertinage, la débauche sont pour ainsi dire à l'ordre du jour dans ces pays encore à moitié sauvages, et le Bétanimène (Malgache de Sainte-Marie), après un séjour de deux ou trois mois au milieu de ce foyer de contamination, pendant lequel il a obéi à ses appétits génériques assez développés, revient à sa terre natale, en apparence indemne comme il en était parti." Lastly, I shall recall the fact that the Europeans at Chinese ports suffer much more severely from syphilis than the natives.

It is obvious that it is not with *peculiarities of race* as

determining a relative immunity from syphilis that we have to do in all these cases. The inhabitants of Iceland belong to the Scandinavian stock, which pays a not inconsiderable tribute to syphilis in Norway, Sweden and Denmark. The natives of Greenland differ in no respect from other Eskimo tribes on the western side of North America, who have had frightful visitations of syphilis. In contrast to the negro population of which we have been speaking, the negroes on the East and West Coasts of Africa suffer from syphilis at least as often and as severely as other nationalities. If there be no mistake in the facts, then we are confronted with a riddle which our knowledge of the conditions of living among these peoples does not enable us to solve.

It has been often alleged that syphilitic infection is of a particularly severe character and follows a very protracted course, when it is the sequel of sexual intercourse between persons of different nationalities ; but, whether that be so, we have not facts enough to generalize upon. On the other hand it is a truth borne out by all observations hitherto, that syphilis finds its widest diffusion and its worst types, *cæteris paribus*, in those countries or among those peoples which are visited by the disease for the first time ; and that circumstance, as we shall see in the sequel, is explained on the ground of neglected hygiene.

§ 23. INFLUENCE OF POLICE SURVEILLANCE.

At all times, and at every point in its area of distribution, syphilis has been the more widely spread and the more severe, the more indifferent has been the attention to *public and private hygiene*, the more *prostitution* has been allowed to go uncontrolled, or the more recklessly the population have given themselves up to "*Venus vulgivaga*" and have borne the effects of syphilitic infection with indifference. It is this that explains the frightful ravages which the disease has wrought, and is even still working, among peoples in the lowest stages of civilisation ; this explains also the wide diffusion and the malignancy of syphilis in every country where there is no regular surveillance of prostitution by the State,—in Algiers, Egypt,

China, Japan, Mexico, Brazil, Peru, and Chili; and it explains further, the increased prevalence of the disease in times of war or in circumstances where the concentration of large masses of people for a considerable period makes surveillance difficult. It is under the latter circumstances, and particularly in regions with a poor and uneducated population wanting the services of a medical profession, that syphilis has not unfrequently broken out and become prevalent in epidemic and endemic form.

Instances of the effect of the movement of troops in war upon the diffusion of syphilis are given by the following writers: by Metzger,¹ of the general spread of the disease in East Prussia in consequence of the invasion by Russian troops; by Boulgakoff,² of its increase in the Government of Tchernigov since the concentration of large bodies of troops there; by Rigler (l. c.), of the increased prevalence of syphilis in Asia Minor since the fourth decade of this century in consequence of military operations; by Olympios (l. c.), of its general diffusion in Greece since the war of liberation; by Lorenz,³ of the remarkable increase of it in Chur at the time of the French occupation in the beginning of the century; and by Ochwad⁴, of the enormous accession of it among the Prussian troops during the Danish campaign of 1864, when the average rose from 29 per 1000 in time of peace to 164 per 1000. How much can be done to diminish syphilis by a strict surveillance of prostitution is shown by the decrease of the disease in the armies of several European states subsequent to the introduction of a more strict control over syphilitic cases occurring among the troops. Thus, in the *British army*⁵ within the United Kingdom, the proportion per 1000 men from 1860 to 1863 (*i.e.* before the passing of the Contagious Diseases Act) was 265; from 1864 to 1869 it was 207; from 1870 to 1879 it was 124. In the *French army*⁶ from 1865 to 1869 it was 106, and 85 from 1872 to 1873; in the *Italian army*,⁷ 120 from 1864 to 1865, and 66 from 1874 to 1876.

¹ 'Verm. med. Schriften,' Königsb., 1788, i, 81.

² 'Bull. des Sc. méd.,' 1824, xxiii, 206.

³ 'Jahresber. der naturforsch. Gesellsch. Graubündens,' Chur., 1868-69, 66.

⁴ 'Kriegschirurg. Erfahrungen u. s. w.,' Berl., 1865.

⁵ 'Army Statistical Report for the year 1879,' Lond., 1881, 11.

⁶ Granier, 'Lyon méd.,' 1880, Nr. 18, 5.

⁷ Sormani, l. c., 226.

§ 24. REMARKABLE EPIDEMICS AND ENDEMICS OF SYPHILIS.

The history of syphilis abounds in other proofs of the decided influence of the social factor upon the amount and character of the disease. I shall limit myself here to stating the facts relating to the previously mentioned outbreaks of syphilis in the character of an epidemic or endemic; these phenomenal occurrences are, from every point of view, most interesting, and they are most important for understanding that great period in the history of syphilis which falls in the end of the fifteenth century and beginning of the sixteenth.

The first observation belonging to this group comes from Scotland in the middle of the seventeenth century; at the time of Cromwell's invasion of the south-western districts of that country, a disease appeared, under the name of *Sibbens* (or *Sivvens*), which was afterwards (1694) carried by troops to the Highlands.¹ Its greatest prevalence was about the middle of the eighteenth century in the south-western counties of Dumfries, Kirkcudbright, Wigton, Galloway, and Ayr; as late as 1825-40, there were sixty cases of it, from Highland districts, admitted into the Glasgow Infirmary, but since that time, the name of the malady has disappeared from Scottish medical writings. Descriptions of the *sibbens* tend to show that we have to do here with severe forms of syphilis, with frambœsia-like exanthems, and probably also with syphilis complicated with other diseases, especially of the skin (scab). It appears that the malady was endemic mostly among the poor, filthy, and neglected inhabitants of certain districts, and that it was spread not only by sexual intercourse, but also by contagion in other ways (wearing clothes in common, sleeping in the same bed, eating and drinking out of the same dish), as well as by heredity.

¹ See: Freer, 'Diss. de syphilide, nec non de morbo Sivvans dicto,' Edinb., 1707; Blair, 'Miscell. Observ. in the Practice of Physik,' Lond., 1718; Hill, 'Cases of Surgery,' Edinb., 1772; Gilchrist, 'Edinb. Med. Essays and Obs.,' new ser., iii; Swediaur, 'Von der Lustsenche,' from the French, 1799, ii, 247; Craigie, 'Elements of the Pract. of Physik,' Edinb., 1836, i, 681; Faye, 'Norsk. Mag. for Laegevid.,' 1842, v, 2; Skea, 'Monthly Journ. of Med.,' 1844, iv, 615; Wills, ib., 282.

Next in time after the sibbens, comes the outbreak of endemic syphilis in Norway and Sweden,¹ which was known as *Radesyge*, or the "bad disorder." The earliest traces of this endemic in Norway may be followed back to 1720; by the middle of the century it had spread more widely, reaching its acme in the last quarter when it was so prevalent, mostly along the coast of the Bergen and Christiansand departments, that special hospitals had to be provided for the reception of the numerous patients. Within the last twenty years, the radesyge has lost its endemic character in that locality. In Sweden the disease first showed itself in 1762, when the Swedish troops came back from the Seven Years' war; so that a connexion between the outbreak and that event may be traced. A second importation of syphilis falls in 1790, at the time of the Swedish troops returning from the war in Finland. During the present century, there has been a considerable decrease of this malady in Sweden; it is still met with oftenest in Bohuslän, especially in the districts of Tjörn, Orust and Lahne, but it has long since lost its endemic character. In the radesyge, as in the sibbens, we have to do with severe forms of syphilis, or that disease complicated with scabies, lupus, and other things; and here again it is a malady confined mostly to the poverty-stricken inhabitants of neglected districts, and spreading by the same kinds of contagion as in the former case, as well as by heredity. From being much confused with *spedalskhed* (leprosy), it was a long time before radesyge was correctly understood; Hebra succeeded in showing that the various

¹ See the following: 'Afhandl. om Radesygen,' Kjöbenh., 1792 (German ed. by Mangor, Altona, 1797); Mangor, 'Underretning om Radesygens Kjendetegn,' ib., 1793; Pfefferkorn, 'Ueber die norwegische Radesyge,' Altona, 1797; Boecker, 'Edinb. Med. and Surg. Journ.,' 1809, Oct., 420; Vought, 'Observ. in exanthema arcticum vulgo Radesyge dictum,' Gryph., 1811; 'Sammandrag af berättelser om veneriska sjukdomar, &c.,' Stockh., 1813; Cederschjöld, 'Inledning till en närmare kännedom om de så kallade urartade veneriska sjukdomarne, &c.,' Stockh., 1814; Holst, 'Morbus quem Radesyge vocant, &c.,' Christiania, 1817; Hedlund, 'Svenska Läk. Sällsk. Handl.,' 1818, v, 176; Hünefeld, 'Die Radesyge, &c.,' Leipz., 1828; Charlton, 'Edinb. Med. and Surg. Journ.,' 1837, July, 101; Hjaltelin, 'Diss. de radesyge,' Kiel, 1839; Hjort, 'Norsk Mag. for Laegevidensk.,' 1840, i, 1; Kjerulf, 'Hygiea,' 1847, xii, 173; Boeck, 'Norsk Mag. for Laegevidensk.,' 1852, And. R., vi, 203; Hebra, 'Zeitschr. der Wien. Aerzte,' 1853, i, 61, 1855, i, 121; Huss, 'Om Sverges endem. sjukdom.,' Stockh., 1852, 10, 33, 43; Broch, 'Le Royaume de Norvège, &c.,' Christiania, 1876, 54.

forms of disease described under that name were secondary or tertiary syphilis, or congenital syphilis, and that lupus and common ulcers of the skin had been included in the general conception of it.

Another relation of the same family is the so-called *Jutland syphiloid*, the origin of which is traceable to importation of syphilis by Russian sailors, or more probably by troops from Sweden or Norway, in the middle of last century.¹ It appears to have been confined as an endemic mostly to the northern part of Jutland, where there were still many cases to be seen during the present century (1837-1842). The attention of the Danish Government was first called to it in 1817, and the report drawn up by v. Deurs supplying the needed information, which tends to show that this syphiloid is in all respects of the same type as the sibbens, the radesyge, and the syphilis-endemics next mentioned.

Completely analogous to these endemics is the *Dithmarsian or Holstein disease*.² The origin of it is traced to the circumstance of a large number of stranger navvies, especially from East Friesland, flocking into the Süder-Ditmarschen for the work of embanking the Crown-Prince dyke; and if they did not introduce the disease, they certainly were the chief occasion of its spreading. By the year 1789, the malady was so prevalent in the marshes and the Gheest that, in some villages, the whole population was affected; towards the end of the century, it showed itself in other parts of Holstein as well, so that, in 1801, the attention of the Government was drawn to it. In 1806 its diffusion was almost universal, reaching at last to Kiel on the east coast; it was not until 1840 that any considerable decrease of this so-called "syphiloid" was remarked, but at the present day it appears to have quite died out as an endemic.

¹ See v. Deurs, 'Jorn. for Med. og Chir.,' 1835, June; Otto, 'Transact. of the Prov. Med. Assoc.,' 1839, vii, 212; Uldall, 'Bibl. for Laeger,' 1842, 337; Ditzel, *ib.*, 1845, ii, 270.

² Brandis, 'Bibl. for Laeger,' 1813, i, 1; Spiering, in 'Hufeland's Journ.,' 1821, liii, Heft i, 64; Hübener, 'De morbi Dithmarsici natura ac indole,' Kiel, 1821; Dührsen, in 'Pfaff's Mittheilungen,' 1832, Jahrg. i, Heft 3 u. 4, 1, and 'Neue Mitth.,' 1835, Jahrg. i, Heft 4, 69; Michaelis, in 'Hamb. Zeitschr. für Med.,' 1842, xxi, 433; Francke, 'Morbus dithmarsicus,' Kiel, 1838; Genters, 'Der morbus Dithmarsicus,' Kiel, 1878.

The *syphiloids of Lithuania and Courland* make another addition to the history of these syphilitic endemics. In the Lithuanian division of East Prussia, syphilis became epidemic in 1757, after the invasion of Russian troops during the Seven Years' war; and it retained its hold as an endemic there down to the first twenty years of this century, when the strict enforcement of police sanitary regulations put an end to it.¹ In Courland the epidemic is said to have broken out in 1800 after the landing of Russian troops on the Dondanga coast. I have no precise information as to the spread of the disease and its duration as an endemic,² except that Adelmann,³ under date 1844, speaks of the general prevalence of very malignant syphilis among the country population around Dorpat.

We come next to certain syphilis-endemics of the same kind on the shores of the Mediterranean,—in Italian and Austrian territory. One of these, known by the name of *Falcadina*, was prevalent in the Venetian province of Belluno from the year 1790.⁴ The disease is said to have shown itself first in the village of Falcade, and to have been imported from the Tyrol or from Fiume; it spread quickly through the mountainous district of Agordo as far as the Tyrolese frontier, but it was not until 1810 that it attracted the notice of the authorities, and about 1830 the endemic was stamped out by means of police sanitary regulations.

Another sickness of this kind, widely known under the name of *Skerljevo* had been observed since the end of last century on the Croatian and Dalmatian coast and for some distance inland.⁵

¹ Theden, 'Erfahrungen aus der Wundarzneikunst, &c.,' Berl., 1782, iii, 9; Metzger, l. c.; Albers, 'Preuss. med. Vereins Ztg.,' 1836, Nr. 22, 23; Schnuhr, ib., 1837, Nr. 50, 51, 1839, Nr. 17, 18, 1841, Nr. 2, 3.

² Tilling, 'Ueber Syphilis und Syphiloid,' Mitau, 1833; Bolschwing, 'Ueber Syphilis und Aussatz,' Dorpat, 1839.

³ 'Med. Ztg. Russl.,' 1844, Nr. 43.

⁴ See Zecchinelli, 'Annal. univ. di med.,' 1820, Marzo, 335; Valenzasca, ib., 1824, Sept., and 'Della Falcadina,' Venez., 1840; Marcolini, 'Memor. med.-chir. di Milano, 1839, 18; Facen, 'Gaz. Med. Lombard,' 1849, 183; Sigmund, 'Zeitschr. der Wiener Aerzte,' 1855, i, 87.

⁵ Boué, 'Essai sur la maladie de Scherljevo,' Paris, 1814; Cambieri, 'Annal. univ. di med.,' 1819, Oct., 5, Dec., 273; Jennicker, 'Oest. med. Jahrb.,' 1819-20, v, Heft 3, 104, Heft 4, 43; Lorenzutti, 'Del male di Scerlievo,' Padua, 1830

According to report, the disease (syphilis) was introduced by sailors or soldiers into the village of Draga near Fiume; the first to take it is said to have been a loose woman named Margaretta (whence the name that it got of *Margaritizza*) who gave it to others, and so laid the beginning of an endemic. From this village it spread along the coast as far as Novi, travelling at the same time inland to Carniola, so that a large district of country was infected within a few months. Notwithstanding most energetic efforts of the Government to combat the sickness by police sanitary regulations, they did not succeed in getting the mastery of it until 1855; and even at the present day syphilis still plays a prominent part in those regions. Belonging to this same endemic was the disease known by the name of *Male di Breno* which was prevalent at the beginning of the century in the commune of Breno near Ragusa.¹

Whereas, in the endemics just mentioned, we have to do not only with syphilis, but with a complex of diseases of various kinds characterised by ulcerations of the skin (scabies, lupus, cancer, scrofula), among which certainly the leading part was always played by syphilis; we meet, on the other hand, with pure endemic syphilis in Servia (under the name of *Frenga*), in Wallachia, Moldavia, Bulgaria, and adjoining regions (under the name of *Bõala*), and in Greece, where it is known as *Spirokolon*. All these were connected in their origin with the events of warfare.²

In Servia, syphilis began to assume an endemic character in 1810, in those districts of the country which were occupied that year by the combined Russo-Servian and the Turkish troops; from them it spread over the adjoining mountainous region on the right bank of the Morawa, and to a less extent over the plains.

It was not until 1844 that accurate information about the disease was obtained, and it remained for Sigmund to give

(Trieste, 1844); Sporer, 'Oest med. Jahrb.,' 1831, Neuste Folge, ii, 211; Michahelles, 'Das Male di Scerlievo,' Nürnberg, 1833; Moulon, 'Nouv. observ. sur la nature . . . du Scherlievo,' Milan, 1834 (1840); Sigmund, l. c., 93, 142.

¹ Id., l. c., 91.

² Concerning *Frenga* and *Bõala*, see Sigmund, l. c., 33, 91. Concerning *Spirokolon*, Olympios, l. c.; Wibmer, in 'Schmidt's Jahrb. für Med.,' 1841, xxx, 305; Pallis, 'Annal. univ. di med.,' 1842, April.

a complete elucidation of its syphilitic nature. The *Böala* is of somewhat later date ; or in other words, syphilis in the corresponding region began to be endemic after the Russo-Turkish war of 1828-29. The endemic syphilis of Greece, known as *Spirokolon*, has the same relations ; it took its origin in the war-years of 1820-25, appearing first in a few eastern districts of Livadia, Bœotia, Locris, and Phocis, and spreading subsequently over other parts of the country.

Another of these syphilis endemics arose out of the events of 1815 in the commune of *Chavanne* (Arrond. Lure, Dep. Haute-Saône) ; the disease was brought by Austrian troops, and it infected the district from end to end within twenty-eight months.¹ Another instance of the endemic occurrence of the malady is in a few localities of the *Circle of Bidschow* (Bohemia),² where “ it has taken deep root, setting at nought year after year all endeavours to eradicate the insidious plague.” Still another case is the epidemic and endemic prevalence of syphilis in the commune of *Capistrello* (province of Abruzzo ulteriore II), of which we have an account by Selli.³

The beginning of the last-mentioned endemic dates from 1859, in which year a woman of the village took to nurse a strange child suffering from congenital syphilis, and became infected by it. Not knowing the nature of the malady, and giving no heed to it in her family intercourse, she soon gave the disease to those about her ; from these it was conveyed to others in the village, and so extensively did it spread that after a lapse of eight years (down to October, 1867, when the attention of the authorities was first drawn to it), during which we hear nothing either of diagnosis or treatment, more than three hundred persons—men and women, children and the aged—were infected with syphilis out of some 3000 who formed the population of the locality.

The development of all these endemics, in which it is with syphilis that we have to do mainly and sometimes exclusively, had taken place principally under the influence of that genetic factor which we started to inquire into in the previous section, namely, *a defective hygiene both public and private*. In all such foci of sickness, the headquarters of

¹ Flamand, ‘ Jorun. complém. du dictionn. des sc. méd.,’ 1819, v, 134.

² Streinz, ‘ Oest. med. Jahrb.,’ 1831, Nst. F., ii, 336.

³ ‘ Annal. du dermatol. et de la syphiligr.,’ 1869, i, 158.

the disease had been those districts or villages which lay remote from the great lines of traffic, which were inhabited by a poor and unintelligent population, indifferent to their manner of life, wanting the comforts of a higher civilisation, and most of all wanting medical supervision and care or possessing only such kind of it as did not suffice for the correct understanding and treatment of the malady. On this point all discriminating observers of the events are agreed;¹ and if any further proof be needed of the decisive importance of that factor in the etiology, we find it in the circumstance that, from the moment the nature of the malady was rightly understood and suitable regulations against it applied with sufficient thoroughness, the disease began to lose its endemic character.

It is further noteworthy that the development of the sickness in many of these endemic centres was started, or at least materially helped, by the concentration of troops, the contingencies of campaigning and things of a like kind tending to aggravate the hardness of living. Again, the spread of the disease occurred not only through sexual promiscuousness, as usual, but very often by other channels of contagion as well, and, above all, in places where the endemic had lasted many years and had deeply infected the population, by way of inheritance. Lastly, the disease developed unusually often into its most malignant forms, in consequence of the want of rational medical treatment.

§ 25. THE ORIGIN OF THE GREAT FIFTEENTH-CENTURY EPIDEMIC.

The mode of origin and the character of these endemics of syphilis appear to me to furnish the key to an understanding of that remarkable episode of the disease in the fifteenth century, an episode which entirely resembles them as regards its type and differs from them only as regards extent. Syphilis had undoubtedly existed in Europe previous to that outbreak, although we cannot now make out the extent of its diffusion. But just at that period there happened a series of events of a

¹ See especially Sigmund, l. c. p. 142.

most unfortunate kind for the social condition of Europe, which led to an increase of the disease both in its area and in its intensity and stamped it with the character of an epidemic. For several years in succession, inclement seasons and floods over large parts of Italy, France, and Germany had brought with them bad harvests, and famine affecting a great part of Europe; and these effects were felt to the most frightful extent in those very years of 1491-95. Severe pestilences, especially plague and typhus, had overrun the south and west of Europe in disastrous epidemics. The corruption of morals had at the same time reached a height which even contemporary writers tell us was without parallel in ancient times. To all these troubles there was added the turmoil of war, which spared no country in Europe, and not only contributed materially to the profound derangement of the social order, but also gave syphilis its chief opportunity of spreading. It was especially Charles VIII's army of mercenaries returning from Italy relaxed by licentiousness, broken up into lawless bands and overrunning France, Switzerland, the Netherlands, and Germany, which carried with them, as we are expressly told by many medical writers and chroniclers of the time, the germs of syphilis over the whole country wherever an adventurous life led them.¹ But the disease must have acquired a still wider range and a greater malignancy than those circumstances gave it, for the reason that practitioners were as yet unacquainted with its nature, their attention being first called to its distinctive characters by the general diffusion which it then reached. So much was this the case that, like typhus, which was likewise spreading universally at the time, it was regarded by them as a malady "of new origin," and was confronted at first with no better weapons than the wise saws extracted from Galen and Avicenna. Just as in the smaller syphilis-endemics of which we have been speaking, so in the severe fifteenth-century epidemic it was the class of low debauchees, the loafers and sots, and those sections of the people hardest pressed by poverty and misery, that supplied most of the

¹ It is probably to this that the disease owes the colloquial names "*morbus gallicus*," "*mala Frantzosa*," and the like, which were then in general use and are so still in many parts of the East.

victims ; and if personages in high places of the State and the Church did not escape, that will not surprise us when we remember that their class had not held aloof from the moral corruptions of the time. Again, as in the more recent endemics, the sickness was sooner or later combated by thorough-going sanitary measures, by an improved hygiene, by the instruction and enlightenment of the people, and by better professional knowledge ; so that at length the victory was gained over the great syphilis-plague of the fifteenth century through the correct recognition of it by the doctors and through the enlightenment of the public. This was the conclusion long ago stated by Benedictus in no obscure words :¹ “ Cur autem tempore isto non reperiantur, diceret quis, gallicantes cum tam saevis accidentibus, sicut apparuerunt ante aliquot annos, et in morbi hujus principiis : ratio est in promptu, quia homines nunc sibi melius cavent ab infectis, vel quia medici docti melius cognoscunt nunc causam morbi, et melius applicant remedia quam tempore anteacto.”

I shall speak of *the relation of syphilis to the so-called Frambæsia (Pian, Yaws, &c.) and to Button-Scurvy*, in treating of those diseases in the chapter immediately following.

¹ Quoted by Luisinus, 172, D. This excellent work of Benedictus, a German physician who practised in Poland, dates probably from the second decade of the 16th century.

CHAPTER III.

YAWS (PIAN), BUTTON SCURVY, AND VERRUGA PERUVIANA.

§ 26. NOSOLOGICAL CHARACTERS OF YAWS.

UNDER such names as Yaws, Pian, and the like,¹ and more particularly under the name of "*Frambæsia*" introduced by Sauvages,² there is understood a peculiar disease of the skin running a chronic course, which was known to the practitioners of former centuries, although it is only in quite recent publications that its nature and geographical distribution have been determined with any degree of precision.

Sketch of the clinical history.—As in the acute exanthemata, there is a premonitory stage, sometimes faintly indicated (Nielen, Rodschied, Mason)³ but particularly well-marked in children, which declares itself in feverishness, broken sleep, general weakness, pains in the limbs, loss of appetite, sometimes even more intense gastric symptoms (Ferrier, Charlouis), and in the dark races, especially the negro, in a peculiar discoloration of the skin, which becomes dull and lustreless at the spots

¹ The words "Yaws" and "Pian," as Mason tells us (see Sauvages, as quoted below), are the colloquial names used in the West Indies for strawberries by the West African negroes and by the natives, the name having been applied to the disease owing to the resemblance of the growths on the skin to that fruit. It is this that led Sauvages to call the malady *Frambæsia* (from *framboise*, a raspberry). The following are some of its other colloquial names: *Buba* or *Boba* in the West Indies, in Brazil, in Timor, and on the Mozambique coast; *Patta* in the West Indies; *Gattoo* at several points on the West Coast of Africa; *Framosi* on the Calabar coast; *Tetia* on the Congo coast; *Momba* in Angola; *Pateh* at various places in the Dutch East Indies; *Amboina pocks* (Bouton d'Amboine) in the Moluccas; *Bobento* in Ternate; *Tonga*, *Dthoke* and *Coco* in the Fiji Islands and New Caledonia; *Lupani* and *Tono* in the Samoa Group; and *Parangi* in Ceylon. In order to put an end to the confusion introduced into medical terminology by the ambiguity of the word "*Frambæsia*," Charlouis proposes to call the disease in question "*polypapilloma tropicum*."

² 'Nosologia methodica,' Cl. x, § 25, Lips., 1797, v, 205.

³ The references to authorities on yaws quoted in the sequel are given in alphabetical order at the end of the section.

where the exanthem afterwards breaks out, and looks as if dusted with flour, from the furfuraceous scaling of the epidermis (Paulet, Levacher, Thomson, Bajon, Milroy ('Report'), Kynsey). These phenomena remit after lasting from eight to fourteen days (Königer, Charlouis), and the exanthem then comes out in the form of small dense nodules, varying in size from a lentil down to a pin's-head; they increase rapidly in size, and, when they are fully ripe, they form resistant nodes from the size of a small nut down to that of a pea. The period of development of the exanthem lasts two or three weeks, seldom a whole month (Nielen); and, in the course of it, the epidermis covering the nodule gets macerated and finally detached from the summit, whereupon a strawberry-like or raspberry-like tumour comes into view, having a granulating surface and secreting a thin, yellowish, ill-smelling moisture which dries into a crust. The disease has now reached its height, if the case is to be a mild and simple one; the nodules, which are painful only under considerable pressure, as when their seat is the sole of the foot or the palm of the hand, remain indolent and unchanged for months. The secretion continues to be formed on the surface, and, if the crust be removed, it soon forms afresh; if the secretion adheres to the tumour, it gradually acquires a conical shape like the crust of rupia (Charlouis). The skin-tissues surrounding the nodule undergo a concurrent alteration; it is only rarely, and under particular circumstances to be afterwards mentioned, that an ichorous disintegration of the tumour sets in, with considerable destruction of the soft parts adjoining, and even of the bones. The swelling and tenderness of the lymphatic glands which occur at the time when the exanthem begins to develop (v. Leent, Charlouis), now disappear; and, apart from an occasional troublesome itching in the affected cutaneous spots (v. Leent, Königer) the patient feels quite well. The secretion on the free surface of the tumours gradually ceases, the nodules become dry and shrivelled, and ultimately fall off, leaving behind on the skin a red spot which disappears completely after a longer or shorter period. The principal seats of the exanthem are the palms of the hands and soles of the feet, the forehead, the corners of the mouth, the lips, the armpits, the neck, the prepuce, the scrotum, and around the anus; but other parts of the integument do not always escape, such as the scalp, the chin, the external auditory meatus, and the skin of the chest and belly; while not unfrequently nodules occur also on the mucous membranes—in the nose, inside the cheeks, on the gums, and in the vulva. In many cases there are only a few nodules formed, but in others it appears that the exanthem is more widely diffused over the body, the nodules being sometimes so close together that several become confluent and make a tumour the size of a small apple. The disease lasts from a few months to a year or more; its course is made especially protracted by the not unfrequent recurrences; and in such cases there will be an opportunity of observing the exanthem in the several stages of its development at one and the same time (Rodschied, Mason, Thomson). The issue of the disease, in these uncomplicated cases where there are

no special injurious circumstances, is always towards cure. On the other hand, it has a less favorable type in feeble children, in whom the extensive spreading of the exanthem—as exanthems usually do spread in children—is very apt to be followed by exhaustion. The type is less favorable, also, in those cases in which suppuration of the nodules, with ulceration of the soft parts and even carious destruction of bone, has been brought about by external irritation, for example, when the nodules are on the sole of the foot; and, again, in patients who are suffering at the same time from some other dyscrasia, such as scrofula or syphilis, or where there has been unsuitable treatment, particularly the application of caustics or the abuse of courses of mercury (Königer, Kynsey). It is doubtful whether the internal organs (lungs, liver, spleen, kidneys) are affected by the disease, as is believed in some quarters (v. Leent); the occurrence of these visceral lesions and the fatal result of them, may be always referred to other diseased states, either existing coincidently with yaws, or appearing intercurrently.

Morbid anatomy.—According to the anatomical examinations of the affected tissue made by Paulet, Ferrier, Charlouis, and Pontoppidan, it appears that in yaws we have to do with a chronic dermatitis proceeding from the papillary layer and extending deeper into the corium in the successive stages of the malady. Charlouis has found, at the beginning of the skin-affection, dilatation and tortuosity of the surface vessels, and afterwards of the deeper, together with escape of colourless blood-corpuscles and extensive accumulations of these in the tissues; and he is convinced that the progressive enlargement of the papillæ of the skin and the changes in the deeper parts associated therewith, have kept pace with the changes in the vascular system. He has found, also, that, as the disease progresses, the hair-follicles, as well as the sebaceous and sweat-glands and the muscles of the skin, are implicated in sympathy. After repeated microscopic examinations of the morbid tissues, he is unable to arrive at the actual cause of these pathological changes. Pontoppidan found, on cutting out a nodule and examining it under the microscope, after it had been hardened in alcohol, that the crust consisted of a conglomerate of dried-up epidermis and pus-corpuscles, beneath which was a stratum of granulation-cells (as in granulosomatous growths), the papillary stratum being entire although somewhat flattened, the rete mucosum, on the other hand, wasted and occupied by round cells, while the corium was free from adventitious elements. He could never discover fungi, such as *Trichophyton* or *Microsporon*, either in the slough or in the layers beneath.

In the opinion of nearly all observers, yaws is a *peculiar and specific infectious morbid process, a disease sui generis, which has nothing whatever in common with syphilis*. The view of the syphilitic nature of yaws, which was started by several of the earlier observers and has found adherents

more recently in Rollet, Grenet, Copland, and Roquete, rests upon errors of diagnosis. Against it there are not only the typical characteristics of the disease as above sketched, which perfectly distinguish yaws from syphilitic diseases of the skin, but more especially the fact that yaws has none of the properties of a constitutional disease, that it has markedly the character of a local malady, that it always ends in complete recovery without medical treatment, and that the use of mercury in yaws has been found to be absolutely injurious. It is further to be observed that both diseases have several times been found together in the same person, each running its course with the phenomena peculiar to it (Levacher, Paullet, Charlouis); and lastly, there is the fact that yaws is especially common among children of from one to ten years of age.

§ 27. HISTORY AND GEOGRAPHY OF YAWS.

The earliest historical notice of yaws¹ occurs in the narrative of travel by Oviedo,² who came to know the disease in Hispaniola (St. Domingo) and who speaks of it under the Spanish name of "Bubas," afterwards in use in Brazil. Next in order of time come medical accounts of the disease from Brazil by Piso and from the East Indies by Bontius, as well as from the West Indies by Labat, who travelled there about the same date (17th century). In later times, the malady was recognised both by medical practitioners and travellers in the tropical parts of Africa and in some of the island-groups of Oceania; and with these extensions the limits of its *geographical distribution* were laid down practically as they exist at the present day.

¹ The disease described by the Arabian physicians (Avicenna, in 'Canon,' lib. iv, Fen. vii, Tract. iii, cap. i, and Ali Abbas, 'Theoric,' lib. viii, cap. 18), under the name "Safat" or "Sahafat," might be inferred to be syphilis rather than yaws. Equally ambiguous and uncertain are all those other notices by mediæval physicians, in which Sprengel ('Beiträge zur Geschichte der Medicin,' iii, 61) would find yaws described and proof afforded of its connexion with syphilis.

² 'Hist. general y natural de las Indias,' lib. ii, cap. 13-14. It is a not unlikely guess that Oviedo's opinion of the American origin of syphilis partly arose out of confounding the latter disease with the "Bubas."

That part of Africa which lies within the tropics forms one of the chief seats of yaws, the principal region, so far as we can judge from the data before us, being the *West Coast from Senegambia down to the Coast of Angola*,¹ together with the adjoining *western parts of the Soudan*,² from which we have more particular accounts of the prevalence of the malady in Timbuctoo and Bornou. On the *northern and north-eastern coast-territories of Africa*, as well as in the *Nile valley*,³ yaws would appear to be of rare occurrence; on the other hand, we again meet with it somewhat frequently in *Madagascar* and the *Comoros*,⁴ and in *Mozambique*.⁵ Its second more considerable region of diffusion is found in several islands and island-groups of the *East Indies*, chiefly the *Moluccas*,⁶ but also *Java*,⁷ *Sumatra*⁸ and *Celebes* (Macassar).⁹ Next in order comes the endemic of it in *Ceylon*,¹⁰ and in several of the island-groups of Oceania, particularly *New Caledonia*,¹¹ *Fiji*¹² and *Samoa*.¹³ On the other hand, it appears to be very rare on the mainland of Hindostan and of Further India. In the exceedingly copious medico-topographical literature of these countries I have found one notice of yaws by Huillet, relating to its rather common occurrence among the Hindoo population of Pondicherry. Charlouis mentions that he had seen two cases of yaws in European children, and all the other notices of it from India likewise relate to merely occasional cases. From Further India, there is not a single reference to this disease

¹ See the accounts by Mason, Boyle, Bryson, Ritchie and Nielen.

² See Peyrilhe, Guyon ('Gaz. de Paris,' l. c.) and Duncan.

³ Guyon ('Mém. de méd. milit.,' l. c.), Baudouin and Farnari have observed a few cases among the Arab population of Algiers. Pruner states that yaws does not occur at all in the basin of the White Nile as far up as 5° N.; he had seen a few cases, however, among natives of Egypt, of Abyssinia, and of the Arabian coast.

⁴ Grenet.

⁵ Roquete, Bourel-Roncière.

⁶ Bontius, Heymann, v. Leent (l. c., 1870).

⁷ Heymann, Waitz, v. Leent (l. c., 1867), Charlouis.

⁸ v. Leent (l. c., 1867).

⁹ 'Arch. de méd. nav.,' 1871, April, 248.

¹⁰ Milroy ('Med. Times,' 1876, and 'Lancet,' 1877, ll. cc.), Kynsey.

¹¹ De Rochas, l. c., 20.

¹² Bennett (who gives the disease as endemic also in the Tonga, Society, and Navigator Islands), Fox, de Rochas.

¹³ Bennett, Turner, Königer.

known to me. The third of the more considerable seats of the malady is the *West Indies*; we have accounts of it from *St. Domingo*,¹ *Jamaica*,² *Barbadoes*,³ *Martinique*, *Guadeloupe*,⁴ *Sta. Lucia*,⁵ and notably *Dominica*;⁶ also from *Guiana*;⁷ and lastly from *Brazil*,⁸ where the disease is found to be equally common in all the provinces of its vast territory. From *Central America* there is only one reference⁹ to it, describing its somewhat frequent occurrence in *Punta Arenas (Costa Rica)*.

§ 28. NATIVE HABITATS OF YAWS.

The opinion held by nearly all the earlier observers, and maintained at the present day by Gama Lobo¹⁰ and v. Leent,¹¹ that the original *habitat of yaws* is to be sought for in *Western Africa*, and that the disease had been carried thence by the importation of negroes into those regions of the tropics where it is now endemic, has been overthrown by the observations that have been made in the *West Indies*, in *Brazil*, in the *East Indies* and in the islands of the *Pacific*. Oviedo's account of its occurrence in *Hispaniola* belongs to the time when the island was first colonised by the Spaniards, and when there could be no question of negro importation; and for that reason Copland's opinion that the disease is of African origin and had been introduced into the *West Indies* has been pronounced by Milroy to be "a radical error." Bontius saw it in the *East Indies* as early as the beginning of the 17th century, and he does not say a single word about importation from outside; it is precisely the African population there, as Charlouis tells us, that enjoy a striking immunity from yaws,

¹ Pouppé-Desportes, Pontoppidan.

² Ludford, Hunter, Sloane, Thomson.

³ Hillary.

⁴ Labat, Savarésy, Ferrier, Paulet.

⁵ Levacher.

⁶ Keelan, Milroy ('Report'), Nicholls, Bowerbank.

⁷ Kunsemüller, Schilling, Nielen, Hille, Pop, v. Leent (l. c., 1880), for *Surinam*; Rodschied, for *Rio Essequibo* (Brit. Guiana), Bajon; Campet, Nissaeus, Segond, Dumontier, for *Cayenne*.

⁸ Corneiro, Sigaud, Rendu, Bourel-Roncière.

⁹ 'Arch. de méd. nav.,' 1864, Nov., 374.

¹⁰ According to Bourel-Roncière.

¹¹ 'Arch. de méd. nav.,' 1880, l. c.

and it is prevalent at certain places in the south-western islands where Africans have never come. In Brazil also, it was already endemic at the beginning of the 17th century, as appears from the account by Piso ; and Sigaud, who is one of those opposed to the doctrine of importation and in favour of the indigenous nature of the disease in Brazil, makes mention of a manuscript preserved in the Royal Library at Rio de Janeiro, and dating from the year 1587, which treats of the yaws in that country. Least of all, as Königer says, can there be any question of the disease having been imported into the Samoa Group, which were till lately cut off from all intercourse with other countries ; and the less so that the natives of these islands speak of yaws as a malady prevalent among them from time immemorial. If we reflect, further, that many regions of the tropics, such as India and Further India, have been little if at all visited by yaws, notwithstanding an extensive negro immigration, we shall be justified in concluding that the native habitat of the disease is as wide as the area of its diffusion.

§ 29. YAWS A DISEASE OF THE TROPICS.

The limits of this area, however, are very closely drawn, as will appear from the above sketch ; and, so far as relates to a strictly endemic prevalence of yaws, its territory is exclusively within tropical latitudes. The disease is therefore an exquisitely tropical one, or, in other words, it is dependent for its origin and continuance upon *tropical climate*. This is so strikingly the case that, while every province of Brazil is a seat of the malady, it is quite unknown in the Argentine Republic, notably in Monte Video and Buenos Ayres (with a temperature like that of Portugal), notwithstanding the large negro population. We cannot decide, on the basis of any facts known to us, in what way this influence of climate tells upon the production of the disease ; it would seem, at any rate, to be an indirect one, for we shall see that there are considerable differences in the amount of the malady among the various races exposed to

the same climatic influences, and that many regions are quite free from yaws notwithstanding their tropical climate.

§ 30. YAWS NOT ACCOUNTED FOR BY DEFECTIVE HYGIENE.

Indirect, also, must be the influence on the production of the disease exerted by an *injurious hygiene*,—by deficient food, damp and filthy dwellings, and the like. The opinions expressed by Levacher, Bryson, Dumontier and others, that yaws is purely the consequence of these disadvantages in the manner of life of the inhabitants, and particularly of that part of the inhabitants most exposed to them, namely, the negroes, is open to the objection that this race is as much subject to these disadvantages in many regions of the tropics which are quite exempt from yaws, as in the actual habitats of the disease; and we may take it to be a radical error of Chassaniol to suppose that the whites would suffer to the same extent as the negroes if they were subject to the same influences of climate and hygiene.

§ 31. CONTAGIOUSNESS OF YAWS—INHERITED AND RACIAL PREDISPOSITION.

There can be no doubt that yaws has underlying it a *specific cause, a morbid poison*. There is, indeed, indisputable proof of this in its exquisite *contagiousness*. On that point all authorities without exception are agreed, and the evidence of it is not only in the clinical observation that those who avoid contact remain exempt from the disease, but also in the results of various attempts to convey the disease of purpose, by inoculating the morbid poison.¹ There are other points which the available data do not enable us as yet to decide: whether, as most of the authorities assume, the virus keeps itself alive solely by continuous reproduction within the bodies of the sick and by successive transmissions, somewhat in the manner of the syphilitic or smallpox poisons, or whether,

¹ See Mason, Milroy ('Report'), Bourel-Roncière, p. 55, v. Leent, 'Arch. de méd. nav.,' 1880, l. c., and particularly Charlouis, p. 460.

under certain circumstances, it may not really originate; and what is the nature of the virus—whether it is a *parasitic* body that we have to do with, as v. Leent¹ conjectures and as the power of reproduction of the poison would seem to show. The disease is also taken to be parasitic by Pontoppidan, but he has not succeeded, as we have already seen, in discovering any fungous elements in the affected parts.

The opinions are divergent as to *hereditary transmission* of the disease (congenitally, at all events, it seems never to have occurred, as there is not a single case of that kind given in the whole literature); Nielen, Paulet, Legoud, Rendu and v. Leent² consider it as proved, while Thomson, Charlouis and Kynsey hold it to be at least doubtful, and Rankine, Mason and Gama Lobo deny it altogether.

On another point we find observers again almost absolutely unanimous, namely, that the susceptibility to the disease differs much among the various *races*. If no race enjoys an absolute immunity from yaws, still the coloured races, and above all the negroes, supply the largest contingent of cases, mulattoes and other half-tints, with creoles, being more rarely affected, and rarest of all the whites. The reason for the exemption of the latter may be in part that they avoid contagion as much as possible; but that is by no means a complete explanation of the fact, as even Mason, the chief exponent of this argument, would admit. “I can recall several white men,” says Ferrier,³ “who have never taken this disease notwithstanding the intimacy of their relations with negresses or mulatto women infected by it;” and that observation carries all the more weight, when we consider that all observers are especially emphatic about the unusual intensity of its contagiousness among the coloured races.⁴

§ 32. ONE ATTACK USUALLY GIVES PROTECTION BUT NOT INVARIABLY.

Wherever yaws has been observed hitherto, it is especially during *childhood* (from the 3rd to the 12th year) that it has

¹ L. c., 1870 and 1880.

² Ib.

³ L. c., p. 54.

⁴ Pruner thinks that the greater development of the papillary processes of the skin in the coloured races may be applied to explain their predisposition to yaws.

occurred. But the often expressed opinion that having survived the disease in youth abrogates the predisposition to it for the rest of life, does not hold good absolutely at all events. Bajon and Thomson had already stated that this removal of susceptibility to subsequent attacks was much the same as in smallpox: that is to say, it was the rule but there were exceptions. Ferrier also speaks of the same person taking it more than once, and Charlouis¹ says: "I can confidently vouch for the fact that framboesia may attack the same person more than once; and the truth of that may be proved not only by inquiring into the history of cases, but also by inoculation."

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¹ L. c., p. 457.

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Button Scurvy of Ireland.

§ 33. CHARACTERS OF BUTTON SCURVY.

A disease of *Ireland*, very like yaws and known by the colloquial name of button-scurvy, formed the subject of a number of papers between 1823 and 1851, by Autenrieth,¹ by Wallace² (who had named it "morula," owing to the likeness of the excrescences on the skin to mulberries), and by Corrigan,³ Osbrey,⁴ Faye,⁵ Patterson,⁶ Wade,⁷ and Kelly.⁸

Clinical characters.—Having been usually preceded for a longer or shorter period by an intense itching of the skin, coming on particularly at night, the disease is ushered in by an outbreak of small round spots, which gradually raise themselves above the skin and grow into tumours in size from a pea to a nut. The colour of these tumours is at first dark red but becomes paler, the epidermis over them becoming at the same time thinner and thinner, and finally disappearing altogether; a granulating surface now protrudes and secretes a serous fluid, which becomes a dry crust on the summit of the tumour, and is quickly reproduced if it be taken off. The excrescence is elastic to the feel, and somewhat painful on pressure; the skin around it shows no kind of morbid alteration. The number of such nodules in the same person varies from one to fifty, or even more. The favourite seats of the exanthem are the palms of the hands, and the inner side of the thighs and arms; more rarely the hairy scalp, and sometimes even the scrotum and perinaum, where it might be very easily mistaken for condylomata. When the tumours have lasted some time they begin to shrivel, the scabs fall off, and disclose a red spot, which shortly assumes the normal colour of the skin. Only in the event of suppurative disintegration of the tumours, which seems to be on the whole rare, is there a cicatrix formed in the skin.

The duration of the malady is usually many months, and it appears to depend as much upon the long persistence of individual nodules as upon recurrences. It is only the exhaustion following a very copiously developed exanthem or one of long persistence that makes button

¹ 'Untersuchungen über die Volkskrankheiten in Grossbritannien u. s. w.,' Tübing., 1823, 132.

² 'Med.-Chir. Transact.,' 1827, xiii, 469.

³ 'Lond. Med. and Surg. Journ.,' 1835, July.

⁴ 'Dubl. Journ. of Med. Sc.,' 1842, July.

⁵ 'Norsk. Mag. for Lægevidensk.,' 1842, v, 16.

⁶ 'Dubl. Med. Press,' 1844, Feb.

⁷ *Ib.*, March.

⁸ 'Lancet,' 1851, Sept.

scurvy dangerous to the health or life; in the great majority of cases the general well-being appears to have been in no wise affected, and symptoms of constitutional disturbance were never observed. Concerning the anatomical structure of the tumours Wade, Corrigan, and Kelly agree in saying that they should be considered as hypertrophic growths of the papillæ of the corium.

The history of this disease is shrouded in impenetrable obscurity. When and where it first showed itself in *Ireland* is not known; this much only is certain, that it has been more rarely seen in recent times than formerly, and it must now be quite extinct if we are to judge from the silence of Irish practitioners about it at the present day. All the authorities above mentioned agree that the disease is certainly not of the nature of syphilis, as was formerly supposed,¹ that it has just as little in common with scurvy,² but that there is discoverable in its type a striking likeness to yaws.³

Button scurvy has been observed mostly in the Southern counties of Ireland, and as a true endemic only in the interior among the country people. There are no doubts among the observers as to its *contagiousness*, whether by direct means or indirect, more particularly by conveyance of the tumour-secretions by means of articles of clothing. A fact stated by Wallace is worth noting in this connexion, that the majority of the patients treated by him were such as obtained their livelihood by dealing in old clothes, by laundry work, and the like. Kelly observed the disease most frequently in shepherds who had either been employed among sheep affected with the scab or had had to do with their wool; and he concluded that button scurvy was a communicated animal disease. Assuming that this observation and the conclusion

¹ The disease was at one time erroneously mixed up with sibbens, the endemic syphilis of Scotland.

² Osbrey says that it is usual in Ireland to speak of chronic skin-diseases as "scurbutic affections."

³ In consideration of the authorities above quoted, including physicians like Wallace, Corrigan, and Faye, I do not feel justified in taking the whole story of this disease for a piece of fancy, or as resting on errors of diagnosis. While one must admit that the statements about it are remarkable and puzzling to a degree, yet they are not more remarkable or more puzzling than those about the verruga-disease referred to in the next sections, the existence of which, with all its peculiarities, cannot longer be rationally doubted or called in question.

drawn from it are correct, the disease of sheep must at all events have been a peculiar one, inasmuch as the scab exists notoriously all over the world, and the conveyance of the scab-mite to man is known to produce quite other effects than those that have been described for button scurvy.

The Peruvian Wart (Verruga Peruviana).

§ 34. HISTORY AND CHARACTERS OF VERRUGA.

In the history of the conquest of Peru, published in 1543 by Zarate, Chancellor of Lima, there occurs a passage which may be translated literally as follows:¹ “ This country, situated between the tropic and the equator, is very unhealthy ; the men here suffer from a wart or small tumour like a boil, very malignant and dangerous, which appears on the face or other parts of the body, and is more destructive than the small-pox and almost as disastrous as the plague itself.” Farther on the chronicler states that the Portuguese soldiers were affected by boils or warts of a very malignant kind, and that not a single person in the army escaped them. Another historian of Peru, who narrates the campaign of Pizarro’s small army, tells us that of the 700 men composing that force, more than one fourth died of hæmorrhages following gangrenous ulcers of the skin. Nothing had been heard in subsequent years of any disease in Peru to which these notices might apply, until Archibald Smith² in 1842, and after him Tschudi,³ Oriosola,⁴ and Salazar,⁵ followed in recent years by Dounon,⁶ Fournier,⁷ Bourse,⁸ and Tupper,⁹ published accounts of a very

¹ Quoted by Dounon, l. c.

² ‘Edinb. Med. and Surg. Journ.’ 1842, July, 67.

³ ‘Arch. für physiol. Heilkde.’ 1845, iv, 378, and ‘Oesterr. med. Wochenschr.’ 1846, 505.

⁴ ‘Gac. med. di Lima,’ 1858, April ; ‘Med. Times and Gaz.’ 1858, Sept., 280.

⁵ ‘Gac. med. di Lima,’ 1860. (Founded on his thesis of 1858.)

⁶ ‘Étude sur la verruga, maladie endémique dans les Andes Péruviennes,’ Par., 1871. (Given also in ‘Arch. de méd. nav.’ 1871, Oct., 255.)

⁷ ‘Arch. de méd. nav.’ 1874, Sept., 156.

⁸ Ib., 1876, May, 353.

⁹ ‘Ueber die Verruca peruviana,’ Inaug.-Diss., Berlin, 1877. (Of some interest for the morbid anatomy only.)

peculiar disease of the skin, occurring endemically within a very limited district of Peru, which was known by the name of "the wart" (verruca). Between this and the severe cases of sickness among the Portuguese troops in the sixteenth century some connexion, they thought, might exist; although it was a noteworthy fact that the verruca is now met with in only a few places on the western slopes of the Peruvian Andes, being quite unknown in the rest of the country or found only in an occasional imported case.

Clinical characters.—The outbreak of the disease is preceded for several weeks by a feverish condition, during which the patient complains of general weakness, want of appetite, headache, giddiness, a cramp-like feeling of contraction in the gullet (very characteristic), and particularly of exceedingly acute pains in the muscles, bones, and joints. When the exanthem begins to appear all these troubles remit considerably, and vanish altogether during its further development. The exanthem comes out in the form of elevated reddish spots as large as a lentil or a pea, which grow to tumours of cylindrical or hemispherical or conical shape the size of a raspberry or filbert up to that of a pigeon's egg; the colour becomes more reddish-blue, the consistence being soft at first (or always if the growth be rapid), but more elastic afterwards (or from the first if the development be slow), and the surface tender. As the tumour increases in extent the epidermis becomes thinner, the surface of the nodule appears to be cleft like a wart, and blood in larger or smaller quantity begins to pour from the fissures and cracks, sometimes without any provocation, but more often in consequence of some pressure or other mechanical irritation, such as lying on an uncomfortable bed. These bleedings are often difficult to stop, and they are sometimes so copious that anæmia quickly ensues. The number of the tumours is, like their size, very various: sometimes there is only one to be found on the patient, which may be an ordinary sized wart; in other cases the patient's body is covered with hundreds of excrescences of various sizes. The exanthem is found most often and most abundantly on the extremities, next to them on the face, on the hairy scalp, and on the neck, more rarely on the sole of the foot or palm of the hand, and rarest of all on the skin of the trunk, where Tschudi did not see it once among the fifty cases observed by him. In many cases warts occur also on the mucous membranes,—on the conjunctiva, in the mouth, nose, pharynx, and larynx, and in the vagina; in these situations they give rise to bleeding likewise, and if we may infer from hæmorrhages occurring by the mouth and anus in the course of the disease, the mucous membrane of the stomach and intestine would appear to be also a seat of the tumours. Whether the morbid product develops in parenchymatous organs as well (liver, kidneys) or, as Tschudi alleges, in the bones, is very doubtful; post-mortem examinations have been made

hitherto in only a few cases of death from verruga, and the information from these is, at all events, not conclusive, the anatomical changes that have been found in the solid viscera seeming to point rather to complications with other diseases such as malarial sickness, dysentery, and pneumonia.

The duration of the disease, or of the tumours in their developing stage, amounts to several months (two to three on an average), unless death had ensued prematurely either from the disease itself or from some intercurrent malady at its commencement or at its height. The issue of the local process may be a gradual drying and shrivelling of the tumour until it falls off from the skin (this is particularly the case with small warts); or it may be purulent disintegration beginning at the base and leading to an ulceration of the affected area of skin, which heals slowly and leaves behind it a deep red spot, often lasting for a long time. The process is the same in the warts of mucous membranes, which differ from those of the skin only in being more flattened,—in such places as the conjunctiva and surface of the tongue where they are continually exposed to pressure. The disease is seldom got rid of altogether, a complete cure occurring only where the exanthem is scanty and the bleeding slight; for the most part there remains over some condition of weakness, due to the anæmia following profuse bleedings, or there are nervous troubles, or dropsy, or the like, the patient never quite recovering under such circumstances. Very profuse hæmorrhage which cannot be checked may cause death suddenly, or death may sometimes occur at the commencement of the disease with typhoid symptoms, as Tschudi would appear to have observed. Under the usual circumstances (of which more in the sequel) the mortality from verruga is from 6 to 10 per cent. of the sick.

Morbid anatomy.—The excrescences, on anatomical examination, are found to be cavernous vascular tumours (with overgrowth and distension of the capillaries and veins), whose wide-meshed reticulum contains at first a small amount of embryonic connective tissue and afterwards an abundance of fibrillar tissue. This growth of connective tissue proceeds either from the papillary layer of the corium or from its deeper layers. The retrogression, or drying and shrinking, of the tumour is preceded by obliteration of the vessels, and the detachment of it which sometimes occurs through suppuration may be referred to the same cause. Of the state of the internal organs in fatal cases of verruga we have only two notices: one by Salazar of a case that ended fatally from dysentery, the other by Tupper of two cases, in one of which death ensued from pneumonia and in the other from smallpox. But there is no conclusion to be drawn from these observations as to any morbid visceral changes belonging to verruga itself.

§ 35. ENDEMIC CIRCUMSTANCES OF VERRUGA.

Verruga occurs exclusively, so far as is known hitherto, among the *Peruvian Andes*, in a few valleys between the latitudes of 9° and 16° S., and at elevations of from 700 to 2500 metres (2500 to 8000 feet). Its headquarters are stated to be Santa Ulaya (prov. Huarichi), Matucana and a few other villages in the province of Cocachacra, the valleys at the foot of the Cerro de Pasco, inhabited by miners, several valleys in the province of Chiquiang and the mountainous districts to the south of Lima. Beyond these limits only imported cases are observed, notably on the littoral of Peru; Tschudi had never seen it in the Sierra or on the eastern slope of the Andes, and it is equally unknown in Chili, Bolivia and New Granada. Under certain circumstances, chiefly, it would seem, in consequence of some considerable concourse of strangers at its endemic centres, the disease sometimes acquires a sort of *epidemic character*. Thus, it is stated by Bourse that an epidemic of that kind developed among the navvies employed in building the so-called Trans-Andean Railway passing right through the verruga-region, as well as among the English engineers who directed the work; the epidemic was declining at the time of his arrival at Callao (beginning of 1874), having lasted for eight months and caused a very considerable mortality among the foreigners.

All the localities affected by the disease have the same *natural features*. They are deep-cleft narrow valleys, whose sides are formed of bare rock (granite and diorite), and their bottom of clay covered with luxuriant vegetation and traversed by a mountain stream which may fall in cascades or flow smoothly. The disease occurs in these valleys just for so far as they continue to be narrow and gorge-like; wherever the valley widens, it may be not more than a few kilometres from the verruga-centre, there is not a trace of the disease to be found.¹ Owing to the conformation of these valleys, the

¹ Tschudi mentions that it may happen to troops marching through that region to be quartered, some of them in Santa Ulaya, and others in the village of San Pedro Mama, only a league (three miles) away; among the former the disease has often broken out to a disastrous extent, whereas those in the latter village have remained quite exempt.

day temperature in them is high (maximum of 95° to 103° Fahr.) notwithstanding their elevation, whereas the nights are cold, so much so that a diurnal range of 25° to 35° Fahr. is not unfrequently observed. In contrast to the rainless coast of Peru, there is here a rainy season (August to November), although the rainfall is certainly not very copious ; the dry season lasts from January to June, and in the transition months (December and July), violent storms blow from the Sierra. The population of the verruga-valleys belong mostly to the Ando-Peruvian race ; they are very sparsely scattered, miserable in circumstances and degenerate in body. Their food consists of fruits, salted flesh, rice, a few esculent roots, and eggs ; their drink is spring-water turbid from the admixture of mineral matters, which is fetched in earthen vessels and allowed to stand until the suspended substances subside.

§ 36. VERRUGA A DISEASE DUE TO A SPECIFIC CAUSE.

It is obvious that the proper *cause of the verruga-disease* is not to be looked for in any one of these conditions, nor in all of them taken together. But it has been at the same time impossible hitherto to discover any other factor which might be brought even remotely into relation with the pathogenesis. The disease is undoubtedly specific, although it has nothing more in common with syphilis, leprosy, yaws and other chronic infective diseases than the *locus affectus* (Dounon, Fournier, Bourse). That we have to deal with some noxious agent acting generally and producing acute effects, may be inferred from the fact that the disease has been observed in animals also (dogs, cats, mules, poultry), and that a very short residence of strangers in one of the disease-centres is sufficient to start the malady. One of the cases given by Dounon is that of a missionary who fell ill of verruga after a thirty days' stay at Matucana ; it has been alleged that it suffices to acquire the disease merely to travel through the affected districts, without delaying in them ; but this Dounon pronounces to be a fable.

§ 37. ETIOLOGY OF VERRUGA.

In verruga, as in many similar cases, it has been supposed that the cause of the disease might be found in certain injurious properties of the *drinking water*; it is not so much the water of the mountain streams as of small springs issuing from the rocks, which has been thought to be capable of inducing the disease. This is the general belief among the natives, and it has found a supporter in Tschudi, who adduces in its favour the circumstance that if travellers abstain from drinking the water of these suspected springs,—the “*aguas de verrugas*,”—they escape the disease altogether, while a single draught of it is said to suffice for the production of the malady. All the more recent observers are decidedly opposed to that view. Apart from the fact that nothing, either physical or chemical, is discoverable in the suspected water which could possibly be blamed for causing the disease, we have the observation of Dounon that he himself, and his whole following, drank of the water from these springs despite the urgent warnings of the natives, without taking any harm; whereas, on his questioning those suffering from verruga, they admitted that they had fallen victims to the disease notwithstanding their avoidance of the spring-water.

It is not denied on the part of anyone that something *specifically detrimental* underlies the verruga; but the source and nature of this injurious influence are enveloped in complete obscurity. Dounon has found nothing in the dejecta of the sick or in the contents of the tumours, which would indicate a *parasitic origin* of the disease; he believes that it is an affair of a *miasma*, like the malarial miasm, and that is also the opinion of Bourse. The *spread of verruga by contagion* is absolutely denied by the natives, and also by Smith. Dounon declines to give a decided opinion on the question, and Bourse thinks that contagiousness is at least doubtful; there is, farther, no unanimity among the observers, as to whether the predisposition of an individual to infection is abrogated by having had the disease once. On the other hand, all authorities agree that, although no *race* enjoys an immunity, the whites suffer much more frequently and much

more severely than the Indians and negroes. No white foreigner, says Bourse, who has lived some time in a focus of verruga keeps free from the disease; thus, the whole of the engineers who were superintending the building of the Trans-Andean Railway took verruga, and at the time of that author's visit to the locality half of them had died of it; of forty sailors who had deserted from a British ship and had gone to work on the railway, thirty died of verruga in the course of seven or eight months. While the mortality from it among the dark races amounts to about 6 per cent., it rises among the whites to 12—16 per cent., and, when it is prevalent as an epidemic, to 40 per cent. When the exanthem occurs in the internal organs the issue is nearly always fatal (94 per cent.).

CHAPTER IV.

ENDEMIC GOITRE AND CRETINISM.

§ 38. HISTORICAL REFERENCES.

In the medical writings of antiquity, there are many indications, in the statements made about tumours occurring in the neck, which may be taken as referring to goitre; but there is not a word said of its endemic prevalence. Their silence is in part explained by the fact that the Greek and Roman physicians could have had few opportunities of glean- ing experience in the localities where centres of goitre existed; but it is accounted for doubtless in part also by the slight interest that they took in the study of epidemic and endemic diseases in general. It may be safely inferred, however, from the statements of Pliny,¹ Vitruvius,² Juvenal,³ and Ulpian,⁴ that there were already endemics of goitre in the Alps in their times. The first half of the mediæval period furnishes no medical account of endemic goitre; a few references to it are to be found in some of the Lives of the Saints (the disease being regarded in those days as a punishment from God)⁵ and it is from that period that we have the stories of

¹ 'Hist. natural.,' lib. ix, cap. 37, § 68, ed. Franzius, iv, 409: "Guttur homini tantum et suibus intumescit, aquarum quæ potantur plerumque vitio."

² 'De architectura,' ed. Schneider, i, 220: "Guttur homini intumescit præsertim apud Æquicolas et Medullas Alpinos" (probably the inhabitants of the Maurienne).

³ 'Satyr.,' 13: "Quis tumidum guttur miratur in Alpibus."

⁴ 'Fragmenta:,' "Tumido gutture præcipue laborant Alpium incolæ propter aquarum qualitatem, quibus utuntur."

⁵ Hinkmar, in his account of the life of St. Remi (lib. viii), states that when a famine threatened in the country around Rheims (5th cent.), the saint caused wheat to be ground for distribution among the poor; but the Kelts, being possessed of the devil, burned the mills, whereupon the saint pronounced this anathema upon them: "Omnes qui hoc egerunt, et qui de eorum germine nati

curing the goitre by touching with the King's hand.¹ Belonging to the end of the 13th century is the account by Marco Polo of its prevalence in Yarkand and other regions of the Central Asian plateau. Then, in the 14th and 15th centuries, come the first medical references to endemic goitre, by Arnoldus Villanovanus² for the province of Lucca, and by Valescus de Tharanta³ for the Comté de Foix. Next we have the account of the disease in the Duchy of Salzburg by Paracelsus,⁴ who was the first to treat the subject with thoroughness on the basis of his own observations; he sketched the conditions for the endemic occurrence of goitre in brief but forcible lines, indicating the relation of goitre to cretinism, and supplying the first trustworthy information of any kind about cretinism itself. A somewhat more copious, but still hardly sufficient wealth of materials for the study of the history of endemic goitre and cretinism is provided by the medical writers and the chroniclers of the 16th, 17th, and 18th centuries. Among these were Münster⁵ for the Vallais, Styria and the Pyrenees; Agricola⁶ for Salzburg, Tyrol and the Val Tellina; Lange⁷ for Salzburg, Tyrol, Styria and the Vallais; Stumpf⁸ and

sunt, fiant viri herniosi et fœminæ gutturosæ." A similar legend is given by Hubert, in the Life of St. Gudula; the Bishop Emebert (7th cent.) delivered an anathema, it is said, upon the wicked persons who had despoiled the tomb of that saint, the curse being that their offspring should all be cripples (*claudicati*) and the women goitrous. Hubert, who was Bishop of Liege at the beginning of the 8th century, adds: "Et permanent hodie mulctati."

¹ See du Laurens, 'De mirab. strum. sanandi vi solis Galliae regibus divinitus concessa,' Par., 1609.

² 'Breviar,' lib. ii, cap. iv, Opp. Basil., 1585, 1190: "Nascitur in gula quædoque passio, quæ botium dicitur . . . fiunt nempe in quibusdam regionibus forte ex natura aëris vel aquarum in quibus quasi omnes mulieres vel viri sunt strumosi, sicut est quædam regio quæ est in comitatu civitatis Lucae, quæ dicitur Cariphiana."

³ 'Philonium,' lib. vii, cap. 31, Lugd., 1490, fol. 338: "Botium est morbus proprius aliquibus regionibus sicut est Savarte in comitatu Fuxi; et hoc est ratione regiminis, aut ratione aquarum frigidarum quas bibunt et est morbus hereditarius."

⁴ 'De generatione stultorum,' Opp. Strasb., 1616, ii, 74; and 'Von offenen Schâden,' cap. xix, e. c. iii, 587.

⁵ 'Cosmographia univ.,' Basil., 1550, v, 1.

⁶ 'De re metallica,' Basil., 1657, 542.

⁷ 'Epistol. med.,' Basil., 1554, i, 43.

⁸ 'Chronik,' Zür., 1586, 588.

Campell¹ for the Rhine valley above the Lake of Constance, Leo Africanus² for the Atlas, Ortelius³ for Styria, Simmler⁴ for the Vallais, Eustachius Rudius⁵ for Tyrol, Felix Plater⁶ (one of the best writers on cretinism during that period) for the Vallais and Carinthia, Foreest⁷ for the Val Tellina, De la Vega⁸ for Peru, Gaye⁹ for Guatemala, Höfer¹⁰ for Styria, Tollius¹¹ for Schemnitz (Hungary) and Styria, Hoffmann¹² for the Harz and for Kronstadt in Transylvania, Mittermayer¹³ for the Pinzgau (Tyrol) and the Riesengebirge, Keyssler¹⁴ for Savoy, Haller¹⁵ for Aigle (Rhône valley) and the Bernese Oberland, Bourrit¹⁶ for Aosta, Saussure¹⁷ for Savoy and the Vallais, Lentin¹⁸ for the Harz, Marsden¹⁹ for Sumatra, and Lange²⁰ for Kronstadt (Transylvania).

Towards the end of the 18th century there appeared the first important work on the subject, that of Malacarne²¹ based on his observations in the valley of Aosta; and therewith the scientific inquiry may be said to have begun. All that we know of the earlier *history of endemic goitre and cretinism* reduces itself to this: that a few centres of the endemic were known to exist, the same that continue, as we shall find, to be seats of the malady to the present day; and that

¹ Gubler, 'Beitr. zur med. Topogr. v. Chur.,' Tübing., 1824, 9.

² 'De totius Africae descriptione,' Lugd. Bat., 1632, Pars ii, 420.

³ 'Theatrum orbis terrarum,' Antw., 1570, 92.

⁴ 'Valesiæ et Alpium descriptio,' Ludg. Bat., 1633, lib. i, 19.

⁵ 'De virtutibus et vitiis cordis,' Venet., 1587.

⁶ 'Prax. med.,' lib. i, cap. 3, Basil., 1625, 80.

⁷ 'Observ. et curat. med.,' lib. x, 242.

⁸ 'Commentarios reales . . . del origen de los Yncas, &c.,' quoted in Barton.

⁹ 'Reisebeschreibung nach Neu Spanien.' From the French. Leipz., 1693, 238.

¹⁰ 'Hercules medicus,' 1655, 43.

¹¹ 'Epistol. itinerar.,' Amstel., 1700, 237, 238.

¹² 'De morbis endemiis,' Hal., 1705, in Opp., Genev., 1748, 203.

¹³ 'Diss. de strumis ac scrofulis Büsgensium,' Erford., 1723.

¹⁴ 'Neueste Reisen durch Deutschland, &c.,' Hannov., 1751, 240, 291.

¹⁵ 'Opusc. academ.'

¹⁶ 'Description des glaciers, &c.' Germ. transl., Zürich, 1786, 210.

¹⁷ 'Voyage dans les Alpes,' Neuchat., 1779-96, i, 421, ii, 389, iii, 25, 89, iv, 452.

¹⁸ 'Memorabilia, &c.,' Gött., 1779, 127.

¹⁹ 'History of the Island of Sumatra,' Lond., 1783.

²⁰ In Richter, 'Chir. Bibl.,' 1785, viii, 500.

²¹ 'Sui gozzi e sulla stupidita, &c.,' Tor., 1789, and 'Lettre sur l'état des Crétins,' Tur., 1789. Reprinted in Frank's 'Delectus opuscul. med.,' vi.

endemic goitre is proved to have occurred as early as the pre-Christian era. But as regards cretinism previous to the 16th century, the history is enveloped in complete obscurity.

§ 39. GEOGRAPHICAL DISTRIBUTION.

The *geographical distribution of endemic goitre and cretinism* at the present day extends over nearly the whole of the habitable globe ; but goitre, or goitre with cretinism, occurs everywhere in more or less narrowly circumscribed spots, and in very marked association with definite conditions of locality. On European soil, the head-quarters are the western and southern slopes of the Alps of Italy, Switzerland and France, the eastern continuations of the chain in Austrian territory, the Pyrenees, the Vosges and the Jura.

Italy.—The endemic prevalence and relative frequency of goitre in *Italy* is shown in a serviceable if not absolutely exhaustive manner in the following table, which I have compiled from the recruiting-lists published by Sormani,¹ showing the men pronounced unfit for service on account of this disease from 1863 to 1876.

The number of persons liable to military service, examined during those thirteen years, amounted to 2,000,000, and of these 42,863, or 20·9 per 1000, were declared to be unfit on account of goitre. The table shows the proportion exempted for goitre in the respective provinces per 1000 conscripts.

Table of Exemptions from Military Service on account of Goitre in the various Provinces of Italy.

Territory.	Province.	Per cent.
Lombardia ²	67·6
	Sondrio	262·7
	Brescia	118·4
	Bergamo	86·7

¹ 'Geografia nosologica, dell' Italia,' Roma, 1881, p. 22, ff. In estimating the results derived from this statistical conspectus, it should not be forgotten that it relates only to the male population over the age of twenty. The statistical data of cretinism cannot be used, inasmuch as cretinism and idiocy are included in the same list.

² See accounts in 'Oest. med. Jahrb.,' 1832, 1839, Nste. Folge, iii, 349, xxi, 3; Balardini, 'Topogr. med. della prov. di Sondrio,' Mil., 1834, 55; Menis,

Territory.	Province.	Per cent.
Lombardia . .	Como	80.8
	Cremona	59.2
	Milano	48.4
	Pavia	26.2
	Mantua	6.9
Piemonte ¹	59.4
	Cuneo	109.8
	Torino	78.1
	Novara	21.9
	Alessandria	21.2
Liguria ²	30.8
	Genova	32.0
	Porto Maurizio	25.9
Veneto ³	17.8
	Belluno	77.6
	Udine	38.1
	Vicenza	20.3
Emilia	5.2
	Modena	14.0
	Reggio Em. . . .	9.4
	Bologna	6.6
	Parma	4.2
Umbria	10.6
	Perugia	18.7
Marche	1.2
Toscana	2.7
	Massa e Carrara	18.7
Roma	0.6
Abbruzzi e Molise	1.9
Campania	2.9

'Saggio di topogr. med. della prov. di Brescia,' Bresc., 1837, i, 134; Guislain, 'Lettre méd. sur l'Italie,' Gand, 1840, 11; Comolli, 'Gaz. med. Lombard.,' 1848, 304; Palcari, 'Annal. univ.,' 1851, Oct.; Strambio, 'Gaz. med. Lombard.,' 1856, Nr. 22; Demortain, 'Gaz. hebdom. de méd.,' 1859, 683; Tacchini, 'Observ. intorno al cretinismo, &c.,' Pavia, 1859; Lombroso, 'Ricerche sul cretinismo in Lombardia,' Milano, 1859; Castiglioni, 'Il cretinismo nello Valtellino,' Mil., 1860; Lussana, 'Studi di cretinismo in Lombardia,' Mil., 1860; 'Relazione della commissione per lo studio del cretinismo in Lombardia,' Mil., 1864.

¹ See: Foderé, 'Essai sur le goître et le crétinisme,' Turin, 1792, Germ. transl. Berl., 1796, 72; Ferraris, 'Giorn. delle sc. med. di Torino,' 1838, 1840, ii, 370, vii, 385; Garbiglietti, ib., 1845, Giugno; Dubini, 'Gaz. med. di Milano,' 1845, Nr. 33, 1847, Nr. 46; Maffoni, 'Atti dell' Accad. med.-chir. di Torino,' 1846, ii, 453; 'Rapport de la commission créée pour étudier le crétinisme,' Turin, 1848; Grange, 'Compt. rend.,' 1850, ii, 58; Guista, 'Gaz. med. delle Stati Sardi,' 1851, Nr. 12; Dallera, 'Giorn. dell' Accad. med.-chir. di Torino,' 1851, xi; Biffi, 'Sul cretinismo nella valle d'Aosta,' 1861.

² Lombroso, 'Ricerche sul cretinismo endemico in alcuni punti della Liguria,' Mil., 1865.

³ Facen, 'Gaz. med. Lombard,' 1851, Nr. 19, 1869, Nr. 21.

Territory.	Province.	Per cent.
Puglie	0.4
Basilicata	2.3
Calabria	1.9
Sicilia	1.3
Sardegna	0.8

The following table shows where the maximum amount of goitre, ranging from 10 to 30 per cent., falls within the smaller areas :

Aosta (Prov. Turin)	317 per 1000
Val Tellina (Prov. Sondrio)	263 "
Crema (Prov. Cremona)	182 "
Saluzzo (Prov. Cuneo)	179 "
Breno (Prov. Brescia)	170 "
Salo " "	166 "
Chiari " "	163 "
Treviglio (Prov. Bergamo)	154 "
Lecco (Prov. Como)	141 "
Cuneo (Prov. Cuneo)	131 "
Susa (Prov. Turin)	124 "
Pinerolo " "	117 "
Clusone (Prov. Bergamo)	116 "

We find accordingly, that the head-quarters of *endemic goitre in Italy* are the district of *Aosta* at the foot of *Mont Blanc*, and the *valleys of the Alpine chain which traverses Piedmont and Lombardy*, particularly the valleys of the *Dora*, the *Po*, the *Adda* and the *Chiese*, and to a less extent the valleys of the *Sesia*, the *Ticino* and the *Adige*. In *Venetian territory* there are considerable centres of goitre in the valleys of the provinces of *Belluno* and *Udine*. The *Mincio* appears to be the boundary towards *Lombardy* of the goitrous region there ; for, while the proportion of goitrous persons on the western shore of the *Lake of Garda* (province of *Brescia*) is 112 per 1000, it amounts only to 4 per 1000 on the eastern shore (province of *Verona*). In like manner the *Po* marks off the endemic goitre of the plain of *Lombardy* from the *Æmilia*; in the districts of *Pavia*, *Lodi* and *Cremona* the number of cases is 30 to 80 per 1000, while in *Voghera*, *Piacenza*, *Parma* and other places, the maximum is only 7 per 1000.

Cretinism also finds its most extensive diffusion in the

same regions of *Italy*. The number of those liable to the conscription who have been pronounced unfit for service on account of cretinism (and idiocy) amounts to 10·7 per 1000 in *Aosta*, 5·9 in *Sondrio*, 5·4 in *Brescia*, and 4·5 in *Chiari*; and it is again the above-mentioned Alpine valleys of Piedmont, Lombardy and Venetia that have the largest number of cases. We also meet with centres of goitre and cretinism, of moderate extent, on the northern slopes of the *Apennine* in Piedmont, Liguria and the *Æmilia*, as well as in a few localities of Umbria and the *Abruzzi*,¹ and at some points in the *Terra di Lavoro*.² In the rest of Central and Southern Italy, the two diseases, and especially cretinism, are far from reaching the importance that they have in the three former regions. In Sicily and Sardinia they do not occur endemically at all, so far as I can judge from the information before me.

According to the returns (very incomplete) collected by the Sardinian Commission in 1845-46, of the diffusion of cretinism in the quondam Kingdom of Sardinia, the number of cretins per 1000 of the population reached the enormous figure of 27·9 in the district of *Aosta*, 2·2 per 1000 in *Cuneo*, 2·5 in *Ivrea* (*Turin*), and 2·1 in *Saluzzo*. For Lombardy, the number of cretins in 1859 was estimated by *Lombroso* at 5000, or 1·7 per 1000 inhabitants, the provinces of *Brescia*, *Sondrio* (*Val Tellina*), *Como* and *Cremona* being most severely affected.

France.—Directly connected with the severe centres of goitre and cretinism in Piedmont is the endemic prevalence of both diseases in the Alpine Departments of *France*. I take the following data on the *distribution of goitre and cretinism in France* from the official report of the Commission of Inquiry drawn up by *Baillarger*.³

The total number of goitrous persons in France above the age of 20, amounted to 370,403; the number of cretins and idiots to about 120,000. Taking the population of the country to be 36,000,000, the proportion of goitre per 1000 was 10·4, and of cretinism and idiocy 3·3. The following table shows the proportion of the goitrous per 1000 inhabitants in the several departments.

¹ Guislain, l. c.

² de Renzi, 'Topogr. e stat. med. della città di Napoli, &c.,' Nap., 1845; Costa, 'Esculapio,' 1840, i, Nr. 6.

³ 'Rapport de la commission d'enquête sur le goître et le crétinisme en France,' Paris, 1873.

Table of Goitre in the several Departments of France per 1000 inhabitants.

	Department.	Per 1000.		Department.	Per 1000.
I.	Savoie . . .	133·7	IV.	Eure . . .	9·0
	Hautes-Alpes . .	111·0		Haute-Vienne . .	7·8
	Haute-Savoie . .	92·0		Seine-Oise . . .	7·7
	Arriège . . .	82·7		Gard . . .	6·6
	Basses-Alpes . .	76·9		Yonne . . .	6·3
	Hautes-Pyrénées .	62·3		Orne . . .	6·2
	Jura . . .	58·9		Somme . . .	5·9
	Vosges ¹ . . .	56·8		Landes . . .	5·9
	Aisne . . .	52·9		Charente . . .	5·7
	Alpes-Maritimes .	50·7		Sarthe . . .	4·8
		73·8		Seine infér. . .	3·8
II.	Loire . . .	49·0		Bouches-du-Rhône .	3·7
	Rhône . . .	46·0		Seine-Marne . . .	3·7
	Puy-de-Dôme . .	44·6		Calvados . . .	3·4
	Haute-Loire . . .	42·8		Eure-Loire . . .	3·3
	Oise . . .	36·9		Gers . . .	3·2
	Drôme ² . . .	36·9		Var . . .	3·2
	Meurthe ³ . . .	33·0		Corse . . .	3·0
	Cantal . . .	32·0		Tarn . . .	3·0
	Haute-Saône . . .	31·6			5·1
	Haute-Marne . . .	30·8	V.	Indre . . .	2·9
III.	Ardèche . . .	29·5		Maine-Loire . . .	2·6
	Isère ⁴ . . .	29·0		Tarn-Garonne . . .	2·4
	Lozère . . .	29·0		Gironde . . .	2·4
	Dordogne . . .	25·0		Vendée . . .	2·3
	Pyrénées-Orient .	24·0		Seine . . .	2·2
	Meuse . . .	22·6		Ille-Vilaine . . .	2·1
	Doubs . . .	22·0		Loiret . . .	2·0
	Saône-et-Loire . .	21·7		Cher . . .	2·0
	Haute-Garonne . .	21·0		Nord . . .	2·0
	Basses-Pyrénées .	21·0		Lot-Garonne . . .	1·6
	Corrèze . . .	20·0		Hérault . . .	1·6
		30·9		Pas-de-Calais . . .	1·6
III.	Ardennes . . .	17·0		Mayenne . . .	1·3
	Aveyron . . .	17·0		Loir-Cher . . .	1·2
	Lot . . .	17·0			2·0
	Ain . . .	16·0	VI.	Indre-Loire . . .	0·8
	Vaucluse . . .	15·7		Vienne . . .	0·7
	Aude . . .	15·0		Loire infér. . .	0·6
	Allier . . .	11·7		Charente infér. . .	0·5
	Côte-d'Or . . .	11·5		Finisterre . . .	0·5
	Creuse . . .	11·0		Deux-Sèvres . . .	0·4
	Nièvre . . .	10·6		Morbihan . . .	0·3
	Aube . . .	10·0		Manche . . .	0·3
	Marne . . .	9·7		Côtes-du-Nord . . .	0·2
		13·5			0·4

¹ See Anzouy, 'Gaz. des hôpit.,' 1859, No. 79.² Saint Lager, 'Deuxième série d'études sur les causes du crétinisme, &c.,' Lyon, 1868, 3.³ Anzouy, l. c., and Ancelon, 'Gaz. hebdomadaire de médecine,' 1857.⁴ Nièpce, 'Traité du goitre et du crétinisme,' Par., 1851.

Of the relative amount of *cretinism* in the several French departments, these returns afford a less certain measure, inasmuch as the data include both cretinism and idiocy. The head quarters, however, of that malady are the departments of Hautes-Alpes and Savoie, with 22 and 16 per 1000 respectively; next to them, as regards frequency of the disease (from 4 to 6 per 1000) come the Basses-Alpes, Hautes-Pyrénées, Haute-Savoie, Isère, Ardèche, Drôme, Alpes-Maritimes, Arriège, and Haute-Garonne; and there are further a few minor centres of cretinism in Aveyron, Lot, Haute-Loire, Vosges, Puy-de-Dôme, Pyrénées Orientales, Oise, Aisne, Meurthe, and Haute-Marne.

Comparing the amount of goitre and of cretinism in the several departments, it will be seen that the frequency of goitre is by no means a measure of the frequency of cretinism; it is only in the departments of Savoy and the High Alps, which are among the worst centres of goitre, that we find at the same time the worst centres of cretinism; whereas in the Jura, which takes one of the highest places for goitre, cretinism is met with to a very slight extent (2·5 per 1000, including idiots).¹

In the department of *Savoy* the head-quarters of cretinism (and goitre) are the valleys of the Maurienne and Tarentaise, with 22·7 and 14·5. cretins per 1000 inhabitants respectively.² In the *Hautes-Pyrénées*, both diseases are most widely spread in the valleys of Luchon, Bigorre and Lavedan.³ The prevalence of goitre is very remarkable in the hilly country of the *Aisne*, especially in the communes of Fouconcourt and Suzy, while the amount of cretinism is at the same time very small.⁴ In the department of the *Rhone*, both diseases are prevalent, particularly in the mountainous parts (St. Laurent and Beaujeu); in the arrondissements of Lyon and Villefranche the number of goitrous persons (in 1851) was 1·5 and 2·4

¹ Monnier, 'Annal. du Jura,' 1853; Moretin, 'Etiologie du goître,' Par., 1854.

² 'Rapport de la commission Sardaigne,' 124; Grange, 'Gaz. méd. de Paris,' 1848, 820.

³ See: Boulinière, 'Itinér. descript. des Hautes-Pyrénées franç.,' Par., 1825, ii, 197; Marchant, 'Observ. . . à l'étude des causes du crétinisme,' Par., 1842; Garrigou, 'Bull. de l'acad. de méd.,' 1868, xxxiii, 715.

⁴ Mahue, 'Gaz. des hôpit.,' 1852, 546.

per 1000 inhabitants, and of cretins 0·4 and 1·0 per 1000.¹ In the *Puy-de-Dôme* it is the southern valleys of the Upper Auvergne that form the chief seats of goitre and cretinism;² in the *Oise*, it is particularly the Nyonnais (arrond. Compiègne) that suffers from endemic goitre;³ for the *Haute-Marne* there are definite accounts of goitre only for Bussièrès;⁴ the *Nièvre* has goitre endemic but is free from cretinism;⁵ in the *Seine-inférieure*, goitre occurs only in the arrondissement of Rouen, being confined to twenty-five villages on the banks of the Seine between Pont d'Arche and Duclair, and in only one family among them were cretins found.⁶

Like France, *Spain* has goitre and cretinism widely prevalent and in considerable frequency, among the valleys of the southern slope of the Pyrenees. The worst localities are the valley of the Aran, and the valleys from Cardous to Ribeira, corresponding to the department of Haute-Garonne on the other side; next to these come Lladore and Ladrons and the valleys of Paillas, d'Estaon, Cinca and Essera. There are also considerable endemic spots of goitre and cretinism in the valleys of the Cantabrian mountains (Asturias and Galicia), in the valleys of the Sierra Morena and Sierra Nevada (New Castile and Estremadura), and in the frontier district between Estremadura and the Portugese province of Alemtejo, in which latter both diseases are likewise indigenous.⁷

Cretinism in Switzerland.—The following data will serve to show the distribution of *cretinism* in Switzerland.⁸ In

¹ Marmy et Quesnois, 'Topogr. et statist. méd. du Depart. du Rhône, &c.,' Lyon, 1866, 103.

² Brieuille, 'Hist. de la soc. de méd. de Paris,' v, Mém., 313; Miral-Jeudy, 'Journ. hebd. de méd.,' 1831, May; Saint-Lager, l. c., 49.

³ Guilbert, 'Étude sur les eaux potables . . . du Nyonnais, &c.,' Par., 1857.

⁴ Lacordaire, 'Préc. anal. du trav. de la Soc. de méd. de Dijon,' 1842, 128.

⁵ Gaudin, 'Du goitre endémique,' Montp., 1869.

⁶ Vingtrinier, 'Du goitre endémique dans le départ. de la Seine-infér., &c.,' Rouen, 1854.

⁷ See Thiéry, 'Observ. de phys. et de méd. de l'Espagne,' Par., 1791, ii, 117; Saint-Lager, 'Études sur les causes du crétinisme, &c.,' Par., 1867, 371.

⁸ For goitre and cretinism in Switzerland, see: Ackermann, 'Ueber die Cretinen u. s. w.,' Gotha, 1790; Troxler, 'Der Cretinismus und seine Formen, &c.,' Zürich, 1836; Demme, 'Ueber endemischen Cretinismus,' Bern, 1840; Meyer-Abrens, in 'Häser's Arch. für die ges. Med.,' 1845, vii, 357, and in 'Rösch's Zeitschr. über den Cretinismus,' 1852, iii, 1; Gosse, 'De l'étiologie du goitre et du crétinisme,' Genève, 1853.

1868 an enumeration gave 3431 cretins in a population of about 2,000,000, or 1·7 per 1000;¹ of these one-seventh occurred in the Vallais. The condition is not more than sporadic or moderately common in the cantons of *St. Gall*,² *Unterwalden* (excepting a small spot of cretinism in the commune of Hergiswyl at the foot of Mount Pilatus), *Schaffhausen*, *Zurich* (excepting the district of Meilen and therein particularly Oetweil and Stäffa), *Thurgau*, *Freiburg* (69 cretins and idiots in 1878 in a population of some 110,000, the district of Broye having 18, Glane 16, and Greyerz 14),³ and *Geneva*. The following is a list of the cantons with endemic cretinism, showing the proportion of cretins per 1000 inhabitants :

Uri . . .	9 per 1000.	Solothurn . .	2·3 per 1000.
Vallais . .	6 „	Vaud . . .	2·1 „
Bern . . .	4·2 „	Aargau . . .	2·0 „
Grisons . .	3·4 „	Lucerne . . .	1·6 „
Glarus . .	3·1 „	Neuchatel . .	1·3 „
Basel . . .	2·7 „	Ticino . . .	1·3 „

In the *Vallais*,⁵ cretinism is commonest in the upper part of the Rhone valley (in Martinach, Fully, Saillon, Sitten, &c.) and in the valleys of the lower part of the canton. In *Uri* it occurs most in the valley of the Reuss (in Silenen, Wasen, Schaddorf, Attinghausen, Altdorf, Seedorf, and Flüelen). In the *Grisons*, where the malady has diminished considerably of late,⁶ the chief spots of cretinism occur in the district of Vorderrhein (Disentis, Somwik, Ilanz, Küstris and other places in the Oberalp valleys); further, in the circle of Thuisis (in Thuisis, Kätzis, and especially Domleschg), in the circle of Maienfeld (Trimmis, Zizers, Ems), and at several points in the Lower Engadine, such as Schuls.⁷

¹ 'Brit. Med. Journ.,' 1868, Oct., 393.

² Meyer-Ahrens, 'Schweiz. Zeitschr. f. Med.,' 1852, 173.

³ 'Rapport . . . sur l'inspection gén. des aliénés dans le canton Fribourg,' Bulle, 1878, 13; see also Berchtold-Beaupré, 'Diss. sur le crétinisme,' Fribourg, 1843, as to the disease in the Gotteranthal.

⁴ Schneider, in 'Berner Vierteljahrscr.,' 1840, and 'Zeitschr. der Wien. Aerzte,' 1845, 97.

⁵ Meyer-Ahrens, in Rösch, l. c.; Fauconneau-Dufresne, 'Revue méd.,' 1846, June; Chatin, 'Compt. rend.,' 1853, i, 652.

⁶ Lorenz, 'Jahresber. der naturforsch. Gesellsch. Graubündens,' 1868-69, 65.

⁷ Meyer-Ahrens, in 'Häser's Arch.,' l. c.; Erlenmeyer, 'Preuss. med. Vereins-

Principal seats of cretinism in *Appenzell* are the communes of Grub, Rehtobel, Reute, Walzenhausen and Heiden; in *Glarus*, the Linththal, Sernfthal and Krauchthal (especially the localities of Engi, Matt, Elm, and Betschwand); in *Basel* the commune of Kleinhüningen¹; in *Vaud* the Rhone valley (especially about Aigle and in Boisnoir), the upland valleys of the districts of Chateau d'Oex and Ormonds (Sepay, Rossinière and Rougemont), and the valley of Broye (districts of Mondon and Payerne).² In the Canton of *Aargau*, the disease is most prevalent in Aarau and Lenzburg in the Aarthal, and near the confluence of the Reuss and Limmath (Altenburg, Windisch, Reuss, Gebensdorf and Vogelsang).³ In the Canton of *Lucerne*, mostly in the districts of Zurseel, Willisau and Entlebuch (notably in Romoos). The cretins in the Canton of *Ticino* in 1855 were distributed as follows: 51 in the district of Bellinzona, 33 in Riviera, 30 in Lugano, and 49 in the remaining districts.

At every place in Switzerland where endemic cretinism is met with, there is also a considerable amount of *endemic goitre*; but goitre is endemic at many places in Switzerland besides these, notably in the Prättigau and Puschlav (Grisons), in the parts of St. Gall bordering the lakes, in the Canton of Schaffhausen, and in Geneva.⁴

The centres of disease in the Grisons and in Lombardy join on to an endemic area of goitre and cretinism in the *Alpine provinces of Austria*.⁵ In the *Tyrol*⁶ we meet with both diseases chiefly in the Innthal and in the Vintschgau; in the Duchy of *Salzburg*,⁷ at a few points in the Salzachthal Ztg., 1854, Nr. 7, and 'Arch. der deutsch. Gesellsch. für Psychiatrie,' 1859, i, 13.

¹ Erlenmeyer, 'Arch.,' l. c. On the Baselland, with about 4000 inhabitants, there are 28 cretins; in Basel town, with 24,000 inhabitants, 64 cretins, of whom 24 are in Kleinhüningen alone (with 465 inhabitants). In the Canton of Basel cretinism is also on the decrease.

² Lebert, 'Arch. für physiol. Heilkde.,' 1848, vii, 516; Account in 'Schweiz. Zeitschr. für Med.,' 1852, 365.

³ Zschokke, 'Annal. der Staatsarzneikde.,' v, 537; Michaelis, 'Skizze der Verbreitung des Cretinismus im Aargau,' Aarau, 1843.

⁴ Coindet, 'Annal. de Chimie et de phys.,' xv, 49.

⁵ Skoda, 'Referat über den Inhalt der Berichte, welche über den Cretinismus in der öster. Monarchie eingelangt sind,' Wien, 1861.

⁶ See Gautieri and Mittermayer, ll. cc.

⁷ Michaelis, in Blumenbach's 'Med. Bibl.,' 1789, iii, 640; Wenzel, 'Ueber den

near Salzburg, at Hallein and Golling and as far up as Werfen, thence laterally to Radstadt and along the Salzach towards St. Johann,¹ then in the Pongau valley (on the southern slopes of the Tannen-Gebirge), in Lungau and in the Pinzgau-Thal (at Niedersill, Mittersill and particularly at Bramberg). The goitrous districts in Gross-Arlthal and the Gastein valley are free from cretinism. In *Upper Austria*² the chief seats of both maladies are the banks of the Danube and the Traun: in the Danube valley, the villages of Engelhartzell, Strudin, St. Nikola, Steyeregg, Sarmingstein; in the Traun circuit, the shores of the lake, the low grounds of the Enns and Traun (Steyer, Losenstein, Garto, Enns), and the marshy Kremsthal. In *Lower Austria*³ also, goitre and cretinism occur mostly in the valley of the Danube (villages of Gross-Pöchlarn, Seisenstein, Krumm-Nussbaum, Wörth and Orading), and, next to it, in the valley of the Leitha, particularly in the district of Sebenstein. Both diseases are prevalent very considerably in *Styria* and *Carinthia*, in the latter to such an extent that about 9 per 1000 of the population are cretins, the province being unable to send a full contingent of troops proportionate to its population.⁴ In *Styria*,⁵ the cretins are 7 per 1000, the worst seats of the malady being the circle of Judenburg (especially the Ennstal and Peltenthal) with 21·3 per 1000, and the circle of Bruck (especially the districts of Pernegg and Märzzuschlag) with 15·4 per 1000, while the circles of Marburg, Gratz and Cilli are less severely affected, the two latter having respectively

Cretinismus,' Wien, 1802; Streinz, in 'Oest. med. Jahrb.,' 1829, Nste. F., i, 45; Knolz, ib., 86, 146; Kirchner, ib., 1835, ix, 395; Hofer, 'Württemb. med. Correspondenzbl.,' 1838, viii, 161; Maffei, 'Der Cretinismus in den norischen Alpen,' Erlang., 1844; Klebs, 'Studien über die Verbreitung des Cretinismus in Oesterreich, u. s. w.,' Prag., 1877.

¹ In St. Johann and two adjoining villages, with a total of 1557 inhabitants, there are 91 cretins, or 58·4 per 1000 of the population.

² Guggen, 'Oester. med. Jahrb.,' Nst. F., 1839, xix, 85; Ozlberger, ib., 1840, xxiv, 265; Schaussberger, 'Oester. med. Wochenschr.,' 1842, 1091.

³ Schaussberger, l. c.; Knolz, 'Oester. med. Jahrb.,' 1846, iv, 228.

⁴ Fradeneck, 'Zeitschr. der Wien. Aerzte,' 1844, i, 440.

⁵ v. Vest, 'Salzb. med.-chir. Ztg.,' 1831, Nr. 46, ii, 337; Waser, 'Oest. med. Jahrb.,' 1836, Nste. F., xi, 349; Weiglein, ib., 1842, i, 278; Pilz, ib., 1848, i, 357, iii, 80, and 'Oest. med. Wochenschr.,' 1846, 293; Tengler, 'Wien. med. Wochenschr.,' 1857, Nr. 11; Köstl, 'Der endemische Cretinismus, u. s. w.,' Wien, 1855.

2·9 and 2·0 per 1000. The valley of the Drave is absolutely free from goitre and cretinism, and that is true also of the Duchy of Carniola.¹ Again, the diseases do not occur endemically in the Alpine region of the South Slavonic provinces of Austria (Croatia and Dalmatia), or, in other words, on the eastern spurs of the Carnic and Julian Alps,² small centres of goitre being found at only two villages in the frontier district of Warasdin.³

A second centre of goitre (here and there also of cretinism), but far inferior in extent to those of the Alpine countries, is met with in the *slopes and valleys of the Carpathians*, as, for instance, in a few mountainous districts of Wallachia and Moldavia,⁴ where cretinism, however, is for the most part merely sporadic. The diseases are endemic in *Transylvania* in the circles of Hermannstadt, Kronstadt⁵ and Bistritz (district of Rodna⁶), as well as at a few localities in the Military Frontier (Banat), such as Orsowa, where goitre alone occurs, and in the mountainous parts of *Bukovina*,⁷ where cretinism is endemic at only one village in the southern division of the country. For *Hungary*,⁸ we have accounts of endemic goitre from the administrative department of Grosswardein (valleys of the Körös and Temes); from the counties of Marmaros, Heves (around Erlau, Bodony and other places), Zips, Gömör, Honth (Schemnitz), Bars (Kremnitz), and Neutra (Altgebirg, Herrenggrund, Utmannsdorf) on the right bank of the Danube; from the counties of Pesth, Raab and Wieselburg; from the mountainous districts of Western Hungary, the eastern spurs of the Norican Alps, the Bakonyan Forest, the county of Eisenburg, and the valleys

¹ Fradeneck, l. c.

² Lambl, 'Zeitschr. der Wien. Aerzte,' 1853, ii, 58.

³ Müller, 'Oest. med. Jahrb.,' 1843, iv, 343.

⁴ Dobronrawow, in 'Hecker's Annal. der ges. Heilkde.,' 1835, xxxi, 341; Barasch, 'Wien. med. Wochenschr.,' 1854, Nr. 52; Champouillon, 'Mém. de méd. milit.,' 1868, Mars, 191.

⁵ Meyr, 'Wochenbl. der Zeitschr. der Wien. Aerzte,' 1861, Nr. 46, 370.

⁶ Müller, 'Oest. med. Jahrb.,' 1843, iv, 344.

⁷ Mayer, 'Diss. de strumis, &c.,' Hannov., 1817; Hampeis, 'Oest. med. Jahrb.,' 1846, iii, 109.

⁸ Zipser, 'Mag. für Pharmacie,' 1826, Feb., 179; Lantz, 'Oest. med. Jahrb.,' 1846, ii, 354; Glatte, in 'Wochenbl. zur Zeitschr. der Gesellsch. der Wiener Aerzte,' 1870, Nr. 38; Saint-Lager, 'Études,' 379.

of the Danube and Drave within the county of Baranya. Throughout Hungary, cretinism appears to be mostly sporadic. The endemic occurrence of goitre in *Galizia*, on the northern slope of the Carpathians, is reported by Rohrer,¹ for the circle of Wadowice (in the Mittelgebirg, from Zywiec and Slein through Makow and Jordanow to the villages south of Myslowice). Kazubowsky² adds to this the statement that the focus of disease has its commencement as far off as Droguila; that the malady is more frequently met with the farther one penetrates into the mountains, as at Myslenice, Stroza, Pcim, Kaszina, and Lubnia, decreasing as an endemic towards the central Carpathian chain and disappearing altogether around Neumarkt; and that, in all these localities, cretinism is indigenous along with goitre.

On the plateau of *Southern Germany* there existed in the earlier half of this century, a considerable centre of cretinism in Lower and Middle Franconia (Bavaria),³ bounded by the Spessart, the Rhöugebirg, the Steigerwald and the Hassberg; but, in recent times, it has diminished materially, being now limited in Central Franconia to Iphofen, a village where it was always most intense, and to the villages of Einersheim, Etzelheim and Hellmitzheim, and, in Lower Franconia, to a few villages around Windsheim and Uffenheim, and to the districts of Kitzingen and Gerolzhofen. Of goitre, there are still considerable centres in Upper Bavaria, in the side valleys of the Inn (near Rosenheim), of the Alz (at Trostberg), of the Salzach (at Burghausen and Titmoning), the Traun, Vilz, Isar, Lech, Iller and Wertach; but there is no definite information as to the extent of these endemics.

In *Württemberg*, goitre and cretinism have attained a much wider diffusion than in Bavaria, although here also there has been a material diminution (of cretinism) in recent years.

¹ 'Oest. med. Jahrbh.' 1845, iii, 353.

² *Ib.*, 1843, iii, 248, 376.

³ See Sensburg, 'Der Cretinismus im Untermain- und Rezatkreise, &c.,' Würzb., 1825; Rosenthal, 'Ueber den Cretinismus, u. s. w.,' Münch., 1839; Hoffmann, 'Einiges über den Cretinismus, &c.,' Würzb., 1841; Stahl, 'Verhandl. der Leopold. Akad.,' xxi, p. i, 329; Vogt, 'Würz. phys.-med. Verhandl.,' 1856, vi, 431, 1858, ix, Sitzungsber. viii; Virchow, 'Gesammelte Abhandl.,' Frankf. a/M., 1856, 891; Majer, 'Bayr. ärztl. Intelligenzbl.,' 1860, Nr. 2, 46; Rüdel, *ib.*, 1882, Nr. 1 ff.

According to the special accounts before me from that kingdom,¹ which do not certainly go farther back than the middle of the present century, the number of cretins in the circles of the Jaxt, Neckar and Black Forest, amounted to 3·8 per 1000 of the population; in the circle of the Danube, again, cretinism occurs only in the village of Langenargen, situated on an absolute level by the Lake of Constance, while goitre exists to a slight extent in a few valleys of that circle (at Blaubeuren and in the valley of the Ach).² The centres of cretinism in the Jaxt circle join on to the above-mentioned endemic in Lower Franconia, and include an important one in the Tauberthal (Mergentheim), as well as considerable spots at Gerabronn, Crailsheim and Ellwangen in the Jaxtthal, at Schorndorf in the lower Remsthal, and at Öhringen, Künzelsau, Gaildorf and Hall, which are respectively in the Roththal, Kocherthal and Bühlerthal. In the districts belonging to the Swabian Alp (Aalen, Neresheim and Heidenheim) the disease occurs only to a limited extent in a few valleys. Goitre is everywhere more or less prevalent where cretinism is, and to such a degree in the department of Hall that, out of 1000 conscripts, 154 were discharged as unfit for service on account of goitre. In the circle of the Neckar, goitre and cretinism have their head-quarters in the Remsthal and at Waiblingen in the Wieslaufthal opening into it; next in order come the various valleys within the communes of Vaihingen, Maulbronn, Brackenheim, Marbach, Backnang and Weinsberg, together with the valley of the Enz; whereas in the valley of the Neckar itself, where goitre is endemic and, in some places, very common, cretinism is for the most part found only in sporadic cases, but in occasional villages to a more considerable extent. In the circle of the Black Forest, goitre and cretinism occur mostly in the

¹ See Riedle, 'Beitr. zur med. Statistik Württembergs,' Tüb., 1834; Plieninger, 'Beschreibung von Stuttgart, &c.,' Stuttg., 1834, 115; Memminger, 'Beschreibung des Königreichs Württemberg,' ix; Kerner, 'Württemberg. med. Correspondenzbl.,' 1839, ix, 202; Höfer, *ib.*, 275; Dürr, *ib.*, 1840, x, 25; Rösch, 'Untersuchungen über den Cretinismus in Württemberg,' Erlang., 1844; Faber, 'Württemberg. med. Correspondenzbl.,' 1858, 220 ff.; Ludwig, *ib.*, 1868, 159.

² Erlenmayer, 'Preuss. med. Vereins-Ztg.,' 1854, Nr. 7, 52; Voetsch, 'Württemberg. med. Correspondenzbl.,' 1866, Nr. 22, 327.

communes of Rottweil, Oberndorf, Sulz and Freudenstadt; next to these, in the Nagoldthal and its side valleys, more rarely and at more circumscribed spots in the valleys of the Neckar and Ammer (in Horb, Rottenburg, Tübingen, and Herrenberg). At the highest points of the Black Forest, neither of the diseases occurs at all. In Sigmaringen, they are both found endemic in a side valley of the Neckar, on the northern slope of the Alp.¹

The extent of cretinism in *Baden* is known to us very imperfectly; certainly there is no reliance to be placed upon the statement² that there were only 490 cretins in 1849 (in See-kreis 54, in Mittelrhein-kreis 24, in Oberrhein-kreis 129, and in Unterrhein-kreis 223); for 260 cretins were counted³ in the commune of Neustadt alone (See-kreis), and in Neudenau on the Jaxt (Unterrhein-kreis) they amount to 20 per 1000 of the population.⁴ The more particular accounts relate to endemic cretinism in Paradies (suburb of Constance), Hornberg, Lörrach and Säckingen (Oberrhein-kreis), to the commune of Offenburg (Elgerswegen on the Kinzig, and Ebersweiher), and to the village of Eschelbronn on the right bank of the Enz (Mittelrhein-kreis); further, to Wiesloch, Mosbach, Neckargemünd, Neudenau, the village of Rosenberg in the commune of Adelsheim (Unterrhein-kreis), Hammer-eisenbach (commune Neustadt, See-kreis), and to a few valleys of the Black Forest.

Weber's⁵ results, collected from the Baden recruiting-lists from 1849 to 1855, afford us a moderately safe means of estimating the amount of *goitre* in that country. According to these, the number of persons declared to be unfit for service because of *goitre* was, for the whole country, 39 per 1000 of those examined. The cases grouped themselves, according to locality, as follows:

I. From communes situated altogether in the plain	. 17.1 per 1000.
II. From communes on the plateau 31.4 "
III. From communes in hilly districts 36.8 "

¹ Heyfelder, in Schmidt's 'Jahrb. der Med.,' 1837, xvi, 90.

² Erlenmeyer, l. c., and 'Arch. der deutsch. Gesellsch. für Psychiatr.,' 1859, i, 14.

³ Rossknecht, 'Mittheil. des bad. ärztl. Vereins,' 1854, 25.

⁴ Guerdau, 'Annal. der Staatsarzneikde,' xi, 599.

⁵ 'Mittheil. des bad. ärztl. Vereins,' 1857, 27.

IV. From communes on the plain, but surrounded by

more or less lofty mountains 44·0 per 1000.

V. From the lower mountain communes 48·0 „

VI. From the high mountain communes 56·8 „

The largest number of goitrous cases (from 5 to 10 per cent. of the men inspected) came from the following communes:

Wiesloch	50·0 per 1000.	Freiburg	60·0 per 1000.
St. Blasien	53·2 „	Rastadt	60·0 „
Triberg	53·2 „	Staufen	61·1 „
Mosbach	53·3 „	Bonndorf	61·3 „
Hornberg	53·6 „	Bretten	61·8 „
Säckingen	53·9 „	Salem	69·7 „
Jestetten	54·1 „	Neckargemünd	70·2 „
Constance	54·5 „	Schönau	70·8 „
Ueberlingen	55·3 „	Wolfach	70·9 „
Heidelberg	57·2 „	Waldshut	75·4 „
Müllheim	57·3 „	Pforzheim	84·8 „
Neustadt	57·9 „	Lörrach	96·3 „
Pfullendorf	57·9 „	Stühlingen	105·2 „

The returns of goitre and cretinism for Alsace and Lorraine belong to the time when these provinces were under French rule. According to a rather old account by Tourdes,¹ there was a somewhat considerable amount of both diseases in Lower Alsace, in the valley of the Rhine, and more particularly in the plain between the Rhine and the Ill; and it was found endemically in the valleys of the Vosges and in two villages of the Canton Schlettstadt.² The following figures were collected in 1852:

Arrond. Strassburg,	in 16 villages,	160 goitrous,	99 cretins.
„ Schlettstadt,	„ 17 „	656 „	26 „
„ Weissenburg,	„ 1 „	8 „	— „
„ Zabern,	„ 4 „	50 „	? „

In that province also there has been noted a gradual diminution of the disease, and the same is true of Lorraine,³ where, according to Allaire⁴ and Richon,⁵ goitre is now endemic in a few villages near Diedenhofen and in the Canton

¹ 'Du goître à Strasbourg,' Strasb., 1854.

² Didelot ('Hist. de la soc. de méd. de Paris,' 1780, ii, 119) had previously called attention to those two centres of goitre and cretinism.

³ Simonin, 'Recherc. topogr. et méd. sur Nancy,' Nancy, 1854, 415.

⁴ 'Mém. de méd. milit.,' 1861, May, 365.

⁵ Ib., 1869, August, 97.

of Metz. According to the official returns published by Baillarger,¹ in every 1000 conscripts there were discharged as unfit for service by reason of goitre or cretinism, as follows :

Depart. Haut-Rhin, 33 per 1000 for goitre, 2·7 for cretinism.

Bas-Rhin,	10·8	„	„	2·7	„
Moselle,	30·5	„	„	3·8	„

In the *Palatinate* there are minor centres of goitre and cretinism at Rheinzabern (14 cretins among a population of 2130), at the village of Glanmühlenbach in the Glanthal, at Hagenbach (13 cretins in a population of 1269) and Neuburg (18 cretins among 1700 inhabitants), both of these in the Canton of Kandel, and at Edigheim (8 cretins among 600 inhabitants).² Cretinism and goitre are found somewhat frequently at other parts of the country besides these, particularly Landau,³ but not in the form of a true endemic malady.

In the *Grand Duchy of Hesse* there are a few centres of both diseases in the valley of the Neckar, in the valleys of the Odenwald and in Vogelsberg; according to the official returns made in 1854, there were 151 cretins and idiots in the whole country, of whom 14 belonged to the province of Rhenish Hesse, 25 to Upper Hesse, and 112 to Starenburg. These data, however, are quite unreliable. Far from trustworthy, also, is the enumeration of cretins made the same year in *Rhenish Prussia*. The subsequent inquiries of Erlenmeyer⁴ have shown as a general result—that goitre is extremely rare in the departments of Cologne and Aix-la-Chapelle; rather more common in the department of Düsseldorf, but still merely sporadic; properly endemic in the department of Treves (where there is even a small spot of cretinism at the village of Russhütte in the circle of Saarbrücken); while the department of Coblenz forms the head-quarters of both diseases. In that department, goitre is mostly indigenous in the circles of Zell, Kochem, St. Goar, Mayen, Ahrweiler,

¹ Vide *supra*, p. 128.

² Erlenmeyer, 'Arch.,' 20; Herberger, in 'Würz. phys.-med. Verhandl., 1852, ii, 270; Hermann, 'Blätter für gerichtl. Med.,' 1882, 128.

³ Pauli, 'Med. Statist. der Stadt Landau,' Land., 1831, 176.

⁴ 'Archiv für Psychiatric,' 1858, i, 97.

Neuwied and Coblenz, all of them riparian. The number of cretins (estimated by Erlenmeyer at 1000 for the whole of Rhenish Prussia) amounts in the department of Coblenz to 168 (under twenty years); of these 104 occur in the circle of Coblenz itself, chiefly at the village of Metternich (municipality of Bassenheim), and on the Rhine island of Niederwörth (municipality of Vallendar), where there are 56 cretins in a population of about 800.

When we come to *Central and Northern Germany*, cretinism disappears almost entirely as an endemic malady. Of endemic goitre there are considerable centres in the department of *Wiesbaden*—in certain valleys of the Rhine, Main, Lahn, Dille, and Aar, as well as on the slopes of the Taunus and the Westerwald.¹ In the recruiting returns from the quondam Arch-Duchy of Nassau for the years 1831-40, the number of those pronounced unfit from the respective districts was as follows per 1000 examined:

Braubach . . . 28	Nassau . . . 15.2
Weilburg . . . 19	Eltville . . . 14
Runkel . . . 17	Montabaur . . . 13
Herborn . . . 16.5	Höchst . . . 12
Königstein . . . 16	Langenschwalbach . 11.3
Dillenburg . . . 16	St. Goarshausen . 11

In the quondam principality of *Hesse* (corresponding to the present department of that name) there are considerable foci of goitre among the spurs of the Rhön mountain, in the valleys of the Werra and Fulda, and especially in the circles of Eschwege, Witzenhausen, and Rothenburg.² It occurs to a still greater extent on the southern slope of the *Thuringian Forest* in the domain of Schmalkalden,³ where cretinism is met with as well as goitre, particularly in the districts of Schmalkalden, Brotterode, and a few villages in the Schleusingen circle and in the Arch-Duchy of *Saxe-Meningen*.⁴ From thence the goitrous zone stretches in a wide sweep over

¹ Ib., 24; Falck, 'De thyreophymat. endemico per Nassoviam et Hessiam Electoralem,' Marburg, 1843, and in 'Casper's Wochenschr.,' 1844, Nr. 8; v. Franque, 'Nass. med. Jahrbücher,' 1859, xv, xvi, 619.

² Falck, l. c.

³ Fuchs, 'Phys.-med. Topogr. des Kreises Schmalkalden,' Marburg, 1848; Kirchhoff, 'Mittheil. des Vereins für Erdkunde,' 1880, 65.

⁴ Rehm, 'Zeitschr. für Epidemiologie,' 1870, Nr. 2.

a number of upland villages in the Eisenach country to Munsbach (Gotha), Sonneberg, and Gräfenenthal, to Hildburghausen and other places in Saxe-Meningen, to Ruhla, and to many localities in the valley of the Saal, and from Rudolstadt to Jena and as far down as Dornberg.¹

A quite isolated little spot of goitre and cretinism is met with at the village of Anraff situated in a deep-cleft valley of the principality of *Waldeck*.² But in the *Harz*³ goitre reaches a considerable diffusion, although the cretinism that used to exist there appears to have now quite disappeared.⁴ Also in the high valleys of the *Erzgebirge*, both on the northern or Saxon side (Annaberg, Schweizerberg, Schneeberg, and the foot-hills in Tarant, the Muldethal, and around Freiberg),⁵ and on the southern or Bohemian side; as well as in the valleys of the *Sudetic Mountains*,⁶ equally among their northern or Silesian spurs and their southern or Bohemian.⁷ In all these regions last mentioned, cretinism occurs only sporadically. The plain of *North Germany* and of the *Netherlands* is quite free from endemics both of cretinism and of goitre.

¹ See Wittich, in 'Baldinger's N. Mag.,' 1785, vii, 114; Loder, 'Observ. quaed. circa strumam,' Jen., 1796; Hoff, 'Der Thüringer Wald'; Thieme, 'Der Cretinismus,' Weimar, 1842; Schwalbe, 'Correspondenzbl. des Thüringer ärztl. Vereins,' 1880, Nr. 5.

² Röhrig, 'Die med.-geogr. Verhältnisse im Fürstenthum Waldeck,' Gött., 1857, 6.

³ Michaelis, Lentin, ll. cc.; Baumgarten, 'Hannov. Annal. für die ges. Heilkde.,' 1837, ii, 90.

⁴ Iphofen had previously called attention to this. How much foundation there may be for Blum's statement ('Hannov. med. Correspondenzbl.,' 1853, No. 20, p. 153) as to the occurrence of cretinism in the mining town of Lautenthal, I am unable to say. Heise's account (ib., 1850, No. 17) of a centre of cretinism existing in the district of Hoya (department of Hanover) is clearly based upon an error of diagnosis.

⁵ Iphofen, Thieme, ll. cc.; Neuhoof, 'Dresdn. Zeitschr. für Heilkde,' 1827, v, 359; Petrenz, in 'Claruss und Radius' wöchentl. Beitr. zur Klinik,' 1833, i, 227; Trautzsch, ib., 1834, iii, 346; Meyer, 'Med. Topogr. von Dresden,' Stollb., 1840, 256.

⁶ Lorinser, 'Preuss. med. Vereins-Ztg.,' 1833, Nr. 12; Haneke, in 'Hufel. Journ.,' 1838, lxxxvi, Heft 5, 77; Preuss, 'Die klimatischen Verhältnisse des Warmbrunner Thales u. s. w.,' Bresl., 1843.

⁷ For the distribution of goitre in the mountainous districts of Bohemia see: Streinz, 'Oest. med. Jahrb.,' 1832, Nst. F., ii, 197, 336, 343, 1834, vii, 16; Cartellieri, ib., 1843, ii, 354; Klebs, 'Allgem. Wien. med. Ztg.,' 1876, Nr. 32-34.

In *Belgium* goitre is found endemically in a few of the southern districts (Condroz, les Fagnes, l'Ardenne, la Famenne, Luxemburg); it is rare in the coast districts and in localities on a sandy formation, with the exception of a few villages in the southern division of East Flanders; while in the district of the "polders," it does not occur at all. Of cretinism not more than 74 cases have been enumerated in Belgium.¹

The amount of goitre in *England* is comparatively large, and it seems that there is more of it in the southern and midland counties than in the northern and mountainous districts. In the south, one of the larger centres is found among the chalk-hills of *Sussex*² (in and around Horsham), and *Hampshire*,³ and in the more elevated parts of *Surrey*,⁴ particularly Haslemere. In the western counties, goitre is endemic at several places in *Monmouth*,⁵ in the Forest of Dean (*Gloucester*),⁶ at Worcester, Stourport, and other places in *Worcestershire*,⁷ in one district of *Cheshire*,⁸ and in many parts of *Wales*.⁹ In the Eastern Counties, there is a considerable centre of goitre in *Norfolk*.¹⁰ It is endemic at Ridgemont in *Bedfordshire*¹¹ and near Beaconsfield in *Bucks*.¹² In the Midlands, it is endemic in *Warwickshire*, in the coal-districts of *Notts*¹³ stretching towards Derbyshire, above all in *Derbyshire*¹⁴ itself (where the condition is so common as to be known colloquially in England by the name of "Derbyshire neck") and in the hilly parts of *Staffordshire*.¹⁵

¹ Meynne, 'Topogr. méd. de la Belgique,' Brux., 1865, 317.

² Inglis, 'Treat. on English Bronchocele, &c.,' Lond., 1838; Manson, *Researches on the Effects of Iodine, &c.*, Lond., 1825.

³ Inglis, Austin, 'Lond. Med. and Phys. Journ.,' 1822, xlviii, 298.

⁴ Austin. ⁵ Holbrook, 'Lond. Med. Repository,' 1817, viii, 288.

⁶ Currie, 'Glasgow Med. Journ.,' 1871, Feb., 153.

⁷ Watson, 'Prov. Med. Transact.,' ii, 194; Addison, *ib.*, iv, 138.

⁸ Moffat, 'Brit. Med. Journ.,' 1870, Sept., 340.

⁹ Reid, 'Edinb. Med. and Surg. Journ.,' 1836, July, 47.

¹⁰ Reeve, *ib.*, 1809, Jan., 31.

¹¹ Blower, 'Brit. Med. Journ.,' 1857, Nov., 924.

¹² Rumsey, 'Prov. Med. and Surg. Journ.,' 1844, June. ¹³ Inglis, Manson.

¹⁴ Prosser, 'Account of Bronchocele, &c.,' London, 1769. Lettsom, 'Mem. of the Med. Soc. of London,' 1792, iii, 489; Manson, Inglis, &c., 'Lond. Med. and Phys. Journ.,' 1825, liii, 49; Wood, 'Mem. of the Philos. Soc. of Manchester,' 1819, viii.

¹⁵ Garner, 'Nat. History of the County of Stafford,' Lond., 1844.

Lastly, from the northern counties, we have information of endemics of goitre at Bolton, [Padiham, Church, and Accrington] in *Lancashire*,¹ in *Yorkshire*² (especially frequent), in some parts of *Durham*³ and of *Westmorland*,⁴ in the lead-mine district of Alston Moor (*Cumberland*),⁵ and in the western division of *Northumberland*.

In *Scotland*,⁶ goitre is much less frequent than in England. The interior of Perthshire⁷ and the east coast of Fife⁸ are given as its chief seats, and there are also centres of it in the southern counties⁹—in the east of Wigtonshire and Kirkudbrightshire, in Dumfriesshire and Roxburghshire, in the west of Berwickshire, in the northern districts of the counties of Selkirk, Peebles, and Lanark, and adjoining parts of Ayr, and in the Isle of Arran.¹⁰ The northern counties of Scotland appear to be quite free from it. From *Ireland* we have no special information about goitre; but, according to Low,¹¹ the disease occurs endemically in a few localities.

Cretinism is very rare in Great Britain, the endemic centre of it in the village of Chiselborough (near Petherton in the south of Somerset) of which we have an account by Norris¹² as late as 1847, being now quite extinct, according to Fagge's information.¹³ According to Blackie, cretinism (as well as goitre) is still found on the east coast of Fife and on the east side of Arran, and there is an earlier notice of its occurrence in the latter locality by Reid.

*Norway*¹⁴ and *Denmark* are quite free from endemic goitre and cretinism. In *Sweden*¹⁵ also, apart from minor centres

¹ Black, 'Transact. of the Prov. Med. Assoc.,' 1837, v, 125.

² Inglis, Low, 'Brit. Med. Journ.,' 1878, June, 29, 1882, Jan., 43.

³ Inglis.

⁴ Watson, Bayers, 'Edinb. Med. and Surg. Journ.,' 1824, Oct., 325.

⁵ Savage, 'Lancet,' 1872, July, 20.

⁶ Account in 'Med.-Chir. Review,' 1825, vi, 243; Reid, l. c.

⁷ Marshall, 'Edinb. Med. and Surg. Journ.,' 1832, Oct., 333.

⁸ Blackie, 'Cretins and Cretinism,' Edinb., 1855, 49.

⁹ Mitchell, 'Brit. Med.-Chir. Rev.,' 1862.

¹⁰ Blackie, Reid. ¹¹ 'Brit. Med. Journ.,' 1882, Jan., 43.

¹² 'Med. Times,' 1848, Jan., 257.

¹³ 'Med.-Chir. Transact.,' 1871, liv, 155.

¹⁴ Broch, 'Le royaume de Norvège, &c.,' Christ., 1876, 55.

¹⁵ Huss, 'Om Sverges endem. sjukd.,' Stockh., 1852, 21; Berg, 'Bidr. till Sveriges med. Topogr. och Statistik.,' Stockh., 1853, 47.

in a few mountainous districts of Westmanlandslän,¹ the occurrence of goitre is limited to the district of Faluh and a few neighbouring villages of Stora Kopparbergslän (Dalarne). In 1867 the whole number of goitrous persons in Sweden was reckoned at 628, of whom 579 belonged to that one district;² in the town of Faluh itself, persons with goitres in 1865 were nearly 7 per cent. of the inhabitants.³

In *Russia-in-Europe* goitre occurs endemically in only a few departments.⁴ A minor centre of the disease exists on the shores of Lake Ladoga, both on the western side around Wiborg and Willmanstrand, and on the eastern side in the valley of the Ojat, especially in two villages on the left bank of the stream (circle of Novaladoga, government of Olonetz) where cretinism also is somewhat common.⁵ Goitre occurs, besides, at a village in the Government of Vladimir, in and around the town of Nishni-Udinsk on the Uda,⁶ to a more considerable extent on the slope of the Ural in the Government of Perm—in the circles of Tscherdün (banks of the Wischera), in Werchoturje (also riparian), Jekaterinburg, Kungur and Krasso-Ufimssk,⁷ and at various places in the main chain of the Caucasus.⁸ In *Siberia* goitre and cretinism are met with in much wider diffusion, especially in the Government of Irkutsk⁹ in the valleys of the Lena and its tributaries (in this Government it was estimated that there were in 1870, 34,000 goitrous persons and 161 cretins in a population of about 366,000, the proportion of the goitrous in some villages being from 12 to 25 per cent.);¹⁰ also in the

¹ 'Sveriges Sundhets-Koll.,' Berättelse, 1858, 10.

² *Ib.*, 1867, 31.

³ Hallin, 'Nord. med. Arkiv,' 1870, ii, 53.

⁴ See Baer, 'Zeitschr. der Wiener Aerzte,' 1860, 170 (also in 'Mélanges biologiques,' ii), and ref. in 'Journ. de la Soc. de Statist.,' 1876, Jan.

⁵ Frank, 'Behandl. der Krankheiten, &c.,' Berl., 1835, iv, 57; Oldekop, 'Med. Ztg. Russl.,' 1858, Nr. 8.

⁶ 'Bericht über den Volks-Gesundheitszustand im russ. Reiche für das Jahr 1856,' 236.

⁷ Heine, 'Med. Ztg. Russl.,' 1857, 244; Berkowski, *ib.*, 1859, Nr. 1; Petuchof, *ib.*, 164.

⁸ v. Seidlitz, in 'Virchow's Arch.,' 1881, Bd. 86, 168.

⁹ Gmelin, 'Reise durch Sibirien,' ii, 282; Ermann, 'Reise um die Erde,' ii, 207; Kruhse, 'Dorpat'er Jahresber.,' 1833, i, 529; Heine.

¹⁰ Ref. in 'Journ. de Statist.,' l. c.

Government of Tomsk on the slopes of the Altai range,¹ and in the Chinese frontier districts beyond Lake Baikal in the circle of Nertchinsk (on Baer's authority).

On the *Continent of Asia*, the headquarters of goitre and cretinism are the northern and southern slopes of the Himalaya. In the western regions of Asia, the two diseases occur endemically only here and there in *Asia Minor*, particularly around Bolat, in the valley of the Kutschuk-Mender, in the neighbourhood of Aidin, in Marsovan, in the upper valley of the Euphrates (north-east from Arabkir), and in Egin.² Syria,³ Arabia,⁴ the table-land of Persia⁵ and Bokhara⁶ are quite free from endemic goitre and cretinism. The zone of severe goitre and cretinism in Central Asia begins in the upper basin of the Indus in the Vale of Kashmir (Balti or Lesser Thibet and Ladak)⁷ and extends through the mountainous parts of the Punjab,⁸ and the provinces of Garwhal and Kumaon⁹ to Nepaul¹⁰ and Bhootan.¹¹ Of the prevalence of the two diseases in the *valleys and plateaus of the Himalaya*, we have more particular information only for the southern slope; although the accounts from Ladak, Nepaul, and Thibet, as well as the occasional references by travellers to the occurrence of goitre in the Mongolian districts of Thian-chan (Mountains of Heaven), leave no room

¹ Uspensky, 'Med. Ztg. Russl.,' 1859, 164.

² Rigler, 'Die Türkei und deren Bewohner, &c.,' Wien, 1852, ii, 246; account in 'Journ. of the Roy. Asiatic Soc.,' vi, 204.

³ Robertson, 'Edinb. Med. and Surg. Journ.,' 1843, April, 247; Tobler, 'Beitr. zur med. Topogr. von Jerusalem,' Berl., 1855, 56.

⁴ Pruner, 'Krank. des Orients,' 323.

⁵ Polak, 'Wien. med. Wochenschr.,' 1853, Nr. 14.

⁶ Burnes, 'Calcutt. Med. Transact.,' 1835, vii, 461.

⁷ Mir-Izzet-Ullah, 'Journ. of the Roy. Asiat. Soc.,' vii, 289, 303; Thornton, 'Gazetteer, &c.,' Lond., 1844; Frazer, 'Journal of a Tour to the Himalaya, &c.,' 349.

⁸ Wilson, 'Med. Times and Gaz.,' 1874, Dec., 692; Milroy, 'Transact. of the Epidemiol. Soc.,' 1865, ii, 157 (relating to the district of Simla).

⁹ Bramley, 'Transact. of the Calcutta Med. Soc.,' 1834, vi, 181; Bell, ib., 457; McClelland, 'Some inquiries in the Province of Kumaon, &c.,' Calcutta, 1835, and 'Sketch of the Med. Topogr. . . of Bengal and the N.W. Provinces,' Lond., 1859, 63.

¹⁰ Bramley, Campbell, 'Transact. of the Calcutta Med. Soc.,' 1835, vii, 1; Brown, 'Ind. Annals of Med. Sc.,' 1859, Jan., 176.

¹¹ Saunders, 'Philos. Transact.' for the year 1789, lxxix, 93; Gray, 'Lancet,' 1877, June, 937.

to doubt that the area of the disease extends also over the northern slopes of the Himalaya proper, and over its side-ranges to the north. There is equally little doubt that cretinism is more or less common at many points of this goitre-area, being intensely endemic at a few places.¹ The endemic influences which determine the occurrence of both diseases on the slopes of the Himalaya make themselves felt in a very marked degree beyond it, in the partly undulating and partly flat and swampy plain known under the name of *Terai*, which covers a breadth of sixty miles from the foot of the range, extending between it and the Ganges and Brahmapootra; this region corresponds to a zone of very intense goitre stretching from Assam, through Rungpeer, Dinajepore, Purneah, Tirhoot,² Mallye,³ the plain of Patna⁴ and Bettiah, along the northern frontier of Oudh⁵ through Gorackpore,⁶ Buraech, and Pileabit to Hurdwar and the borders of Rohilkund.⁷ In this region also, cretinism is endemic at occasional points.⁸

Fayrer estimates the number of goitrous persons in the Terai at 10 per cent. of the population. In the district of Tirhoot, Macnamara treated 23,000 goitres in three years, and Cunningham treated 20,000 to 25,000 in Gorackpore during the cold season of 1854-55 (Milroy, Mouat).

There is a third region of goitre in Hindostan on the plateau of Ramagar, Chota Nagpore, Sirgooja, and Sumbulpoor, forming the boundary between Bengal and Gondwana and stretching away towards Orissa. In the mountainous districts adjoining Orissa, we meet with cretinism as well as goitre⁹. In the medical accounts from the *Deccan*, from the *Nilghiri Hills*, from the *Eastern and Western Ghâts*, and from

¹ M'Clelland, Wilson.

² Evans, 'Transact. of the Calcutta Med. Soc.,' 1832, iv, 246; Milroy, l. c.

³ Tytler, 'Calcutta Tr.,' iv, 375.

⁴ 'Report of the Dispensaries in the Bengal and North-Western Provinces &c.,' Calcutta, 1843, a. v. O.

⁵ Greenhow, 'Ind. Annals of Med. Sc.,' 1859, July, 435.

⁶ Mouat, ib., 1857, April, 436.

⁷ M'Clelland, 'Sketch,' 112; Fayrer, 'Lancet,' 1874, Oct., 580, 617.

⁸ Evans, M'Clelland, Fayrer.

⁹ Breton, 'Transact. of the Calcutta Med. Soc.,' 1830, ii, 245; 'Indian Annals of Med. Sc.,' 1858, July, 508.

the *Bombay and Madras Presidencies* generally, there is not a word said of the occurrence of goitre and cretinism. On the other hand, we have information of endemic goitre in the Galle district of *Ceylon*,¹ and of both goitre and cretinism in the mountainous parts of *Burmah* and *Cochin China*,² and on the central table-lands of *Java* and *Sumatra*³ (districts of *Lepoetie* and *Toelang-Bawang*, and particularly the *Aboeng* country). In *China* the two diseases appear to be endemic chiefly in the northern provinces; in *Staunton's 'Account of Lord Macartney's Embassy to China'*⁴ we find mention made of the great frequency of both maladies in *Tartary*; *Morache*⁵ speaks of them as occurring in *Pekin* and in the adjoining valleys; and *Dudgeon*⁶ states that goitre is very often met with in *Northern China*, both on the plains and in the mountains.

Whether goitre and cretinism be endemic in *Australasia* I am unable to decide for certain; the only statements with which I am acquainted are those by *Polack*⁷ and *Thomson*,⁸ who agree that goitre is quite unknown in *New Zealand*, and of *Bennet*⁹ to the effect that it occurs sporadically in *Tahiti*. The absolute silence of all other observers in these regions should warrant us in concluding that endemic centres of goitre are nowhere found there.

Little being known of the disease-conditions generally in the central parts of *Africa*, there is accordingly little known of the occurrence of goitre and cretinism in particular; but what little we do know is not without interest for the etio-

¹ *Bennet*, 'Ceylon and its Capabilities,' Lond., 1843; *Pridham*, 'Historical . . . Account of Ceylon, &c.,' Lond., 1849.

² *Thorel*, 'Notes méd. du voyage d'exploration du Mekong et du Cochinchine,' Par., 1870, 171; *Beaufils*, 'Arch. de méd. nav.,' 1882, April, 291.

³ *Marsden*, 'History of Sumatra, &c.,' Lond., 1783, 42; *Heymann*, 'Krankheiten der Tropenländer, &c.,' Würzb., 1852, 178; Account in 'Arch. de méd. nav.,' 1867, Oct., 250; *ib.*, 1877, Feb., 81; *v. d. Burg*, 'De Gencsheel in Nedel.-Indie,' Batav., 1882, i, 81.

⁴ 'Account of Lord Macartney's Embassy to China,' Ger. Trans. Berl., 1790, ii, 171.

⁵ 'Annal d'hyg.,' 1870, Jan., 55.

⁶ 'Glasgow Med. Journ.,' 1877, July, 331.

⁷ 'Manners of the New Zealanders,' Lond., 1840, ii, 98.

⁸ 'Brit. and For. Med.-Chir. Review,' 1854.

⁹ 'Lond. Med. Gaz.,' 1832, ix, 629.

logical inquiry. Like the coast regions of all other parts of the world, those of the African continent, and the river-basins adjoining them, are entirely free from endemic goitre and cretinism; these include *Lower Egypt*,¹ the *Abyssinian Basin*,² the *East and West Coasts*³ and the littoral of *Algiers*.⁴ On the other hand, goitre is endemic on the *Abyssinian Plateau*,⁵ in a few localities in *Sennaar*,⁶ on the slopes and in the valleys of the *Atlas* (e.g. in *Kabylia*),⁷ in the mountainous parts of *Morocco*,⁸ and, to a very considerable extent in the *Basin of the Niger*.

The earlier accounts, by Mungo Park and Caillé,⁹ of goitre on the slopes of the Cong mountains and in the upper basin of the Niger (Bamera and Bambook in the Kankan country) have been supplemented by the lately published information of Quintin,¹⁰ whose own observations relate specially to Segou-Sicorro, while he adds the general statement that goitre is widely endemic in the valleys of the Greater Soudan. From the islands lying off the African coast we have intelligence of minor centres of goitre in the mountainous interior of *Madagascar*¹¹ and in the *Azores*;¹² on *Madeira* the disease is very rare.¹³ Whether cretinism also is indigenous in those parts, or what its extent may be, does not appear from the accounts; the French authors are quite silent on the point, while Quintin explicitly states that he had not seen a single case of it in the Soudan. It is only in the notice about goitre in Madagascar that there is any mention made of cretinism.

¹ Pruner, l. c., 323.

² Courbon, 'Observ. topogr. . . . sur le littoral de la mer rouge, &c.' Par., 1861, 35.

³ Daniell, 'Sketch of the Med. Topogr. of the Gulf of Guinea,' Lond., 1849, 114.

⁴ Guyon, 'Gaz. méd. de Paris,' 1845, 690; Bertherand, 'Médecine et hyg. des Arabes,' Par., 1855, 409, and others.

⁵ Blanc, 'Gaz. hebdom. de méd.,' 1874, 349.

⁶ Brocchi, 'Giorn. d'osserv. . . . in Egitto, &c.,' Bassano, 1843, v, 597.

⁷ Baudouin, 'Gaz. méd. de Paris,' 1838, 771; Bertherand, 'Mém. de méd. milit., lii, 115; Finot, ib., lvi, 36; Challan, 'Gaz. méd. de l'Algérie,' 1868, 117.

⁸ Leo Africanus, l. c.

⁹ 'Journ. d'un voyage à Tombouktou,' Par., 1830.

¹⁰ 'Extrait d'un voyage dans le Soudan,' Par., 1869, 46.

¹¹ Blumenbach, 'De generis hum. varietate nativa,' Gott., 1795, 261.

¹² Bullar, 'A Winter in the Azores, &c.,' Lond., 1841, ii, 331.

¹³ Heineken, 'Lond. Med. Reposit.,' 1824, xxii, 15.

Our information on the endemic occurrence of goitre in *North America*¹ belongs for the most part to the early years of this century and is very fragmentary. Proceeding from north to south, we first meet with considerable centres of goitre in *Hudson's Bay Territory*² (below 52° N.), on the banks of the Saskatchewan, and at the head-waters of the Elk and Peace rivers, where Richardson found many cases of goitre among the children of the native Indians. We come upon it next in *Lower Canada*, on the banks of the Lorenzo, between St. John and Montreal, and, in *Michigan State*, in the Detroit district on Lake St. Clair. From New England there are accounts of the occurrence of endemic goitre in *Vermont*³ (Counties of Bennington and Chittenden, especially at some points on the Connecticut River), and in *New Hampshire*,⁴ where the disease has been mostly found also on the banks of the Connecticut. In *New York State* there exist (or did exist)⁵ centres of goitre in Oneida County and on the banks of the Mohawk and Genesee; in *Pennsylvania* they occur in Alleghany and Susquehanna⁶ Counties, and on the banks of the French Creek, Sandusky, Monongahela, Big Beaver, and Muskingum. Other notices of endemic goitre relate to the mountainous parts of *Maryland*, to *Virginia* (particularly to Morgantown, the principal place in the coal-bearing county of Monongalia, and to the banks of the Monongahela), to the town of Vincennes (*Indiana*) on the Wabash, to the mountainous districts of *North* and *South Carolina*, to the northern counties of *Alabama*⁷ where there is a good deal of it, and to the district of De Soto (*Louisiana*⁸),

¹ See Barton, 'Abhandl. über den Kropf. . . . in verschiedenen Theilen von Nord-Amerika, u. s. w.' From the Engl., Gött., 1802, and Gibson, 'Philad. Journ. of Med. and Phys. Sc.,' 1820, i, 47.

² Richardson, in Franklin's 'Narrative of a Journey, &c.,' Lond., 1828, 116; Simpson, 'Narrative of a Journey round the World,' Lond., 1847.

³ Mease, 'Observations on Goitre;' Dorr, 'New York Med. Repos.,' 1806, x, 141; Brown, 'Amer. Journ. of Med. Sc.,' 1847, July, 111.

⁴ Mease.

⁵ Denny ('Philad. Journ. of Med. and Phys. Sc.,' 1825, N. S., i, 47) says that in the town of Pittsburg, which used to have a bad reputation for goitre, no new cases have occurred since 1806.

⁶ Later information is given by Smith, in the 'Transact. of the Pennsylv. State Med. Soc.,' 1858.

⁷ Taylor, 'Transact. of the Alabama State Med. Soc.,' 1854.

⁸ Gibbs, in Fenner's 'South. Med. Reports,' ii, 190.

particularly the banks of the Bayou Pierre. Cretinism does not appear to be at all common except at a few points in all this region; at any rate it is stated by Barton¹ that cases of it are rarely met with in the United States. Brown speaks of its occurrence in the valleys of Vermont; in Kneeland's² account of the health of *Massachusetts* (for which State I have been able to learn nothing of the occurrence of goitre), it is stated that there are at least 1200 idiots and cretins in a population of about one million. Praslow³ also has observed somewhat frequent cases of cretinism among a tribe of Indians living near Cape Mendocino, in *California*, as well as among the Spaniards in the mountainous parts of Southern California.

It is in the upper basin of the Rio Grande del Norte (New Mexico) that the great zone of goitre and cretinism begins; a zone which extends with increasing intensity along the Cordillera through Mexico, Central America, and South America as far as Chili, and forms a region of the disease worthy to be named besides those of the Alps and the Himalaya. The occurrence of endemic goitre in the mountainous parts of Mexico had already been pointed out by Gage,⁴ later accounts⁵ confirming the fact and placing the head-quarters of the disease in the territory of Colima on the western slope of the Cordillera, and in the mountain-districts of Tobasco and Chiappas. From Chiappas the endemic is continued direct into *Guatemala*,⁶ in the "tierra templada" of which there are whole villages afflicted with goitre, and thence onwards through *San Salvador*,⁷ *Nicaragua*,⁸ and *Costa Rica*.⁹

In the countries hitherto mentioned cretinism does not appear to occur; for San Salvador its occurrence is positively

¹ L. c., 122.

² 'Amer. Journ. of Med. Sc.,' 1851, April, 349.

³ 'Der Staat Californien, &c.,' Gött., 1857, 64.

⁴ 'New Survey of the West Indies,' Lond., 1699, 236.

⁵ Heller, 'Wiener Sitzungsber.,' 1848, Nr. 3, 122; Matthieu de Fossey, 'Le Mexique, &c.,' Par., 1857, 581.

⁶ Gage, Bernoulli, 'Schweiz. med. Ztschr.,' 1864, iii, 100.

⁷ Dunlop, 'Travels in Central America,' Lond., 1847; Guzman, 'Essai de topogr. méd. de la république du Salvador,' Par., 1869, 124.

⁸ Bernhard, 'Deutsche Klin.,' 1854, Nr. 8.

⁹ Schwalbe, 'Arch. für klin. Med.,' 1875, xv, 344.

denied by Guzman. On the other hand, we meet with a very considerable endemic of it in *New Granada*, which is also the chief seat of goitre. According to data before us¹ both diseases occur there throughout almost the whole valley of the Rio Magdalena, from Neyva in the "tierra fria" downwards through Santa Fé de Bogota, Maraquita, Honda, and other districts as far as the plain of Pinto, at the confluence of the Cauca and Magdalena. Neither in the lower basin of the latter river nor in the parallel valley of the Cauca, nor in the mountainous province of Antioquia between the two, do goitre and cretinism occur; but we again come upon them in endemic form in the basins of the Meta and Apure.

The enormous prevalence of goitre in some of those districts may be judged of from the fact that Foote found hardly a single person in Maraquita who was not the subject of it more or less. It is remarkable that it did not happen to this observer to find a single case of cretinism; whereas there can be no doubt, from the statements of Humboldt and Roulin, and from an earlier account by Caldas,² that this disease also is endemic in the Magdalena valley.

In *Venezuela*, goitre occurs on the plain between Caracas and Valencia,³ and in the mountain range which stretches from Barquicimeto, by Truxillo, Merida and La Grita, as far as Pamplona and the frontier of New Granada.⁴ The basin of the Orinoco is said by Humboldt to be free from goitre, and there is no mention either of it or of cretinism in the numerous medical reports that come to us from *Guiana*.⁵

From New Granada the zone of goitre is continued along

¹ Brandin, 'De la influencia de los diferentes climas del universo sobre el hombre, &c.,' Lima, 1826 (on the distribution of goitre and cretinism in South America generally); Restrepo, 'Memoria que el Secretario de Estado . . . presentó al primero congreso constitucional de Colombia, &c.,' Bogota, 1823; Humboldt, 'Journal de physiol.,' 1824, iv, 109; Roulin, 'Revue méd.,' 1825, iv, 138; Boussingault, 'Annal. de chimie et de phys.,' xlviii, 41; Foote, 'Amer. Journ. of Med. Sc.,' 1852, Jan., 278.

² 'Semenario del nuevo Reyno de Granada,' 1816, 148.

³ St. Iager, 430 (following Dubreuil).

⁴ Roulin.

⁵ Hille (in 'Casper's Wochenschr. der Heilkunde,' 1836, No. 36) says that it did not happen to him to see a single case of goitre during a residence of many years in Surinam.

the Cordillera through Quito, Cuenca and Loxa (in *Ecuador*),¹ and thence onwards by Caxamarca, Huamacucho, Huanuco, and Pasco in the valley of the Hualaga, or in other words, through the central valleys of *Peru*² and particularly the sierra-valleys of the provinces of Libertad and Ayacucho. Cretinism, as Archibald Smith explicitly states, is not indigenous there, and Tschudi makes no mention of it. In *Bolivia* the chief seats of endemic goitre are the provinces of Yungas and Ayopaya. From *Chili*³ we have information of its endemic prevalence around San Felipe, Santiago, and other places. But it is on the eastern slope of the Cordillera, in the *States of the Argentine Republic*,⁴ that goitre finds its widest diffusion—in the provinces of Salta, Jujuy, Tucumana, Los Lueles, La Rioja (particularly the Famatina valley), and Mendoza, in a few districts of the provinces of Cordova and San Luis, and at many places in the States of Corrientes and Entre Rois. As regards the coexistence of cretinism with it, I find only one notice by Mantegazza, who mentions it along with goitre in the province of Salta. We enter on a farther extension of this great area of goitre in the river-basins of the mountainous parts of *Paraguay*,⁵ and in the goitre-region of *Brazil*⁶ which includes a great part of that country, more especially the southern and central provinces of Rio Grande, Santa Catarina, San Paulo, Goyaz (both the western and eastern slopes of the Sierra Geral, in Natividad

¹ Humboldt.

² Smith, 'Edinb. Med. and Surg. Journ.,' 1842, July, 66; Tschudi, 'Oest. med. Wochenschr.,' 1846, 698.

³ Gilliss, in 'U. S. Naval Astron. Expedition to the Southern Hemisphere, &c.,' Washingt., 1855; Guézennec, 'Arch. de méd. nav.,' 1864, juill, 22; Duploux, ib., août, 108; Guyon, 'Gaz. méd. de Paris,' 1862, 345.

⁴ Smith, l. c.; Brunel, 'Observ. topogr. et méd. faites dans le Rio de la Plata,' Par., 1842, 47; Guyon, l. c. (on goitre in Mendoza); Mantegazza, 'Lettere med. sulla America meridionale,' Milano, 1863, ii, 208, 227; Lemos, 'Revista med.-quir. de Buenos Ayres,' 1877, abstract in 'Jahresbericht über Medicin,' 1878, i, 336.

⁵ Mantegazza; Masterman, in 'Dobell's Reports,' 1870, i, 382.

⁶ Sigaud, 'Du climat et des malad. du Brésil,' Par., 1844, 162; Gardner, 'Travels in the Interior of Brazil,' Lond., 1864; Rendu, 'Études topogr. et méd. sur le Brésil,' Par., 1848, 101; St. Hilaire, 'Voyage aux sources du Rio Negro,' Par., 1848, ii, 72; Castelnau, 'Expédition dans les parties centrales de l'Amérique du Sud,' Par., 1850; Tschudi, 'Wien. med. Wochenschr.,' 1858, 423; Schwarz, 'Zeitschr. der Wien. Aerzte,' 1858, Nr. 37, 580.

Conceição, Arrayas, Villa-Boa, and the upper basin of the Parahyba), and Minas Geraes (districts of Barbacena, Ouro-Preto, Sabara and others). In other words, it extends over the whole country, excepting the coast territory and the alluvial plains. According to the express statements of Rendu, St. Hilaire, Castlenau and Tschudi, cretinism does not occur endemically anywhere in Brazil; and it is so rare in its sporadic form that Dr. Faivre (as reported by Rendu) who lived for a long time in the central provinces (the head-quarters of goitre), and had travelled through them many times, had never seen but one case of cretinism.

Of the occurrence of goitre and cretinism in the West Indies I can find no trustworthy information.

§ 40. INSTANCES OF DECREASE OR INCREASE IN RECENT TIMES.

The outline-sketch above given of the geographical distribution of goitre and cretinism applies, generally speaking, only to more recent times. A comparison of it with the state of matters in former periods, in so far as the history enables us to follow it, brings out fluctuations in the amount of one or both diseases, either in the way of *diminution or even entire disappearance of the endemic*, or in the way of *more decided prominence or increase*.

Thus it had been observed by Fodéré¹ for Piedmont as early as the beginning of this century, that "the number of the goitrous and of the absolutely cretinous has diminished within a few years, a fact which travellers who had visited these valleys twenty years ago, and may now revisit them, can easily convince themselves of." This holds good to a still greater extent for more recent times, as appears from the report of the Sardinian Commission and the statements of Dubini. Some fifty years ago a decrease of cretinism was observed in many parts of Switzerland²—in Lucerne, St.

¹ L. c., 189.

² Mayer-Ahreus, in 'Rösch's Zeitschr.,' iii, 7, 15.

Gall, Schaffhausen and the Vallais; and more recently we have accounts to the same effect from Chur¹ and Basel.² In the Pyrenees, as Boulinière remarks, there has been a diminution of the endemic goitre and cretinism within the present century; and the same is asserted for the department of Puy-de-Dôme by Aguilhon,³ and by Anzouy for certain valleys of the Meurthe and the Vosges.

According to Durand,⁴ goitre has gradually disappeared from the valleys of Larboust and d'Oueil, where there were still many cases in 1820. Simonin and Allaire are agreed as to the decrease of goitre in recent times in Lorraine; in a few communes of the Canton Briey (near Metz), where goitre used to be endemic, it has disappeared since 1789.⁵ In the Department of Bas-Rhin also, according to Tourdes, a considerable diminution in the endemic goitre and cretinism has been observed. In Rheims, where goitre was formerly very common, the disease occurs now only in isolated cases,⁶ and the same holds good for Luzarches in the Department of Seine-et-Oise.⁷ The number of cretins in Middle Franconia has declined considerably of late; whereas, in 1860 they numbered 47 at Iphofen, the chief seat of the endemic, in 1880 the number had fallen to 12.⁸ On the other hand, in Lower Franconia (especially around Gemünd), a slight increase of the endemic has been noted.⁹ From the statistics of Rösch¹⁰ it follows that, within the last thirty years, cretinism has declined considerably in several parts of Würtemberg (Weinsberg, Herrenberg, Gerabronn, Waiblingen, and, more recently, in Schorndorf),¹¹ while there has been a small increase in certain communes of the administrative districts of Oberndorf, Horb, Rottenburg and Tett nang. In Thuringia, a few villages of the valley of the Ilm wherein

¹ Lorenz.

² Erlenmeyer, 'Archiv,' 13.

³ 'Gaz. méd. de Paris,' 1851, 135.

⁴ 'Union méd.,' 1851, Nr. 32.

⁵ Pascal, 'Compt. rend.,' 1842, ii, 225.

⁶ Maumené, 'L'Institut,' 1850, Nr. 870, 282.

⁷ Hahn, 'Compt. rend.,' 1869, lxi, Nr. 16.

⁸ Majer, Riedel, ll. cc.

⁹ Vogt, 'Würzb. Verhandl.,' l. c.

¹⁰ L. c., 130.

¹¹ Faber, l. c.

goitre and cretinism used to be endemic, have become quite free from both diseases recently,¹ and in Schmalkalden cretinism has been considerably less common of late.² This is true also of the Harz, where it can hardly be said now that cretinism is endemic. In Chiselbrough (Somerset) the endemic of cretinism, of which we have accounts as late as 1847, is now quite extinct (see p. 142). The Government of Perm has experienced a decrease in the number of goitrous persons within the last twenty or thirty years; on the other hand, the endemic of that disease on the banks of the Lena, as well as in the Government of Irkutsk generally, has sprung up since the Russians took possession of the country.

As early as the beginning of this century, Barton tells us, a decrease of goitre had been observed in certain parts of the United States; and this statement is confirmed by Denny, who says that, in Pittsburg at the time of the first French settlement, goitre was unknown, that it became common in the years following, so much so that in 1798 there were 150 goitrous in a population of 1500, that it remained at this height until 1806, when it began to decline gradually. Wotherspoon³ also states that goitre used to be very frequent around Kent in Maine, but has become a good deal less common since the first thirty years of the century.

All the observers who have written of goitre in Granada⁴ agree in saying that the endemic, which was originally in the "tierra templada," had spread towards the plateau about the end of last century or the beginning of the present, that it has penetrated as far as the "tierra fria," and has made such enormous strides in some places where it was formerly quite unknown (*e.g.* Maraquita in the upper basin of the Magdalena), that only a small part of the whole population escape it. According to Gardner's statements, the disease has assumed an endemic character in the district of Natividad (Goyaz, Brazil) since the first thirty years of this century; and Sigaud in like manner says of the southern provinces of Brazil:

¹ Hof., l. c., i, 22.

² Kirchhoff.

³ 'Statist. Reports of the U. S. Army,' Philad., 1856, 28.

⁴ Restrepo, Humboldt, Roulin.

“Le goître endémique dans les provinces de Saint-Paul, de Sainte Catherine et de Rio-Grande-du-Sud s’enracine chaque jour davantage . . . Il y a vingt ans que cette maladie était à peine connue dans le Rio-Grande-du-Sud : aujourd’hui on l’observe dans les villes de Rio-Pardo, de Cocheira, etc.” In Salta (Argentine Republic) the appearance of goitre, according to Lemos, dates from the arrival of the first Spanish settlers).

§ 41. EPIDEMIC OUTBREAKS.

One of the most interesting phenomena of the kind is the *breaking out of goitre in the form of an epidemic*, or, in other words, the more or less abrupt occurrence of cases of goitre within a relatively short space among a section of the population who had been hitherto exempt from the disease. By far the larger number of cases of that kind have occurred in France, and, it would appear, within the present century (only one of the French accounts dating from last century). At all events the occurrence has been only rarely noted outside France ; but in all cases whatsoever the special external conditions under which the sickness has developed *en masse* are the same.

The first account¹ from *France* of a so-called epidemic of goitre (the only one from last century) belongs to the years 1783—1789, and relates to Nancy. In a regiment which had been quartered for five years at Caen and was moved to Nancy in 1783, 38 men became goitrous in the winter of 1783-84, in the following year 205, in the next thereafter (1786) 425, then 257 in 1787, after that 132 in 1788, and finally 43 in 1789 ; being a total of 1100 men out of 4 battalions. At the same time, not a case of goitre occurred among the troops that had been garrisoned in the town for some time previously (excepting a few cases in a cavalry regiment), nor among the civil population of the town. Further, in this as in almost all the other epidemics among the military, the

¹ I have taken some pains to make as complete a collection as possible of the facts about these so-called epidemics of goitre, but the following sketch does not by any means claim to be absolutely exhaustive.

disease was strictly confined to the common soldiers, the officers, sergeants, and corporals remaining exempt.¹ From Lorraine we have only one account, relating to Pfalzbourg where goitre broke out in 1820-21, being confined to the garrison as before.² Next we have Rullier's account³ (and Percy's) of an epidemic outbreak of goitre in a boarding-house near Paris; under similar circumstances the disease was prevalent in 1815 in the college at Strasburg, to such an extent that more than one-third of the pupils suffered from it.⁴ At Colmar in the autumn of 1859, 109 men of the cuirassiers became affected with goitre;⁵ there was a recurrence in the spring of 1861, again among the cavalry solely; while in 1863 there were cases first among the infantry and afterwards in the cavalry.⁶ At Neu-Breisach there were five epidemics of goitre from 1847 to 1871;⁷ 23 cases in 1847, 27 cases in 1853, 24 cases in 1858, several cases in 1861, and finally an outbreak in 1870 in which the number of those affected in a single infantry regiment of 1002 men reached the enormous figure of 647. From Belfort we have intelligence of epidemics in the years 1876-79; in the first of those years cases occurred only among the pupils of the Lycée, in the following year in the garrison as well and to a very considerable extent, in the next two years occasional cases, and in the last year (1879) also in a boarding-house for women.⁸ In Autun, also, goitre has been epidemic several times among the pupils attending the schools.⁹ In the garrison of Besançon, 10 men became affected with goitre in 1840, and 77 more from 1842 to 1853; in the summer of 1863 the malady broke out among the troops more widely.¹⁰

¹ Valentin, in Simonin's 'Recherch. topogr.,' 411.

² Cheron, 'Mém. de méd. milit.,' 1822, xii, 79.

³ 'Dict. des scienc. méd.' (en 60 voll.), xviii, 549.

⁴ Fodéré, 'Journ. complém. des sc. méd.,' 1829.

⁵ Hansen, 'Mém. de méd. milit.,' 1864, Jan.

⁶ Gouget, ib., 1863, Sept., 180.

⁷ See: Chambé, 'Gaz. méd. de Strasb.,' 1864, Nr. 11; Lanel, 'Mém. de méd. milit.,' 1859, July, 2; Tellier, ib., 1860, July 5; Müller, ib., 1871, March, 244.

⁸ Viry et Richaud, 'Gaz. hebdom. de méd.,' 1881, 457, 480.

⁹ Guyton, 'Journ. des conn. méd.-chir.,' 1852, 386.

¹⁰ Gérard, 'Mém. de méd. milit.,' 1853, xii, 241, 1854, xiii, 152; Artigue, ib., xiii, 1; Saillard, 'Essai sur le goître épidémique,' Par., 1865.

At St. Etienne it became epidemic in 1864 among the soldiers' children;¹ in 1873 an epidemic sprang up among the troops, affecting 280 out of a total strength of 1400;² and 38 additional cases occurred the next year.³

These epidemics have been observed, especially often at Clermont-Ferrand and at Briançon. At the former place in 1822, 50 of the seminary pupils became affected within a few days;⁴ in the garrison many cases occurred year after year from 1843 to 1846 (as well as mumps in the latter year), and again in 1848 and 1850-52; in 1860 there was a considerable epidemic there, and in the garrison of the adjoining town of Riom at the same time; and this happened again in 1862 and in 1880.⁵ The number of cases in each year ranged from 40 to 80. From Briançon we have accounts of goitre being epidemic in the garrison in 1812, 1819, 1826 (134 cases), 1841, 1842-50, 1857-60 (cases especially numerous in the latter part of this period), and in 1863.⁶ In 1863, the malady was prevalent at one and the same time in the garrisons of Montdauphin and Embrun.⁷ The last French epidemic that I have to mention was in the garrison of Annecy (Thonon) in 1866, when 60 cases occurred among 582 men.⁸

Out of France the epidemic occurrence of the malady after this fashion appears to have been observed very rarely. For the year 1820 there is an account from Silberberg (Silesia) of an outbreak *en masse* in a battalion lately arrived, so that only 70 out of 380 men escaped having the dis-

¹ Brisson, 'Mém. de méd. milit.,' 1864, Oct.

² Michaud, 'Gaz. méd. de Paris,' 1874, 17, 67.

³ Utz, 'Mém. de méd. milit.,' 1876, May, 209.

⁴ Nivet, 'Documents sur les épidémies qui ont regné dans l'arrondissement de Clermont-Ferrand de 1849—1864,' Par., 1865.

⁵ See: Nivet, l. c., and 'Revue méd.-chir.,' 1852, Dec.; Fleury, 'Gaz. méd. de Paris,' 1861, 510; Morelle, 'Mém. de méd. milit.,' 1862, Dec.; Halbron, ib., 1864, Feb.; Thibaud, 'Du goître épidémique,' Par., 1867; Chouet, 'Mém. de méd. milit.,' 1881, July, 353.

⁶ Haberkorn, 'Essai sur le goître épidém. considéré dans l'armée,' Strasb.; 1864; Chevalier, 'Mém. de méd. milit.,' 1830, xxix, 323; Gérard, l. c., Collin, ib., 1853, xii, 261 and 1861, July, 1; Larivière, ib., 1859, July, 7; Pastoret, ib., 1, Rozan, ib., 1863, x, 343.

⁷ Rozan, l. c.; Hedoin, ib., 1864, June.

⁸ Worbe, ib., 1867, Feb., 104, Oct., 273, Nov., 369.

ease.¹ It was epidemic among the pupils of the Pauline Charity at Stuttgart in 1824 and 1833, 16 children out of 39 being affected in the former year, and 39 out of 47 in the latter.² In the Russian campaign against Turkestan in 1877, there were 245 cases of goitre among the 2753 troops who captured and held the town of Kokaun, and it became necessary to alter the location of the garrison.³ Sigaud⁴ gives an account of the sudden appearance of goitre among Brazilian recruits in Rio-Urubú (Goyaz); so severe was this outbreak that the recruits took to flight in terror and hastened to their homes in the province of Para.

All these so-called epidemics of goitre have the following peculiarities in common:—(1) Excepting the cases at Nancy and Paris, they occurred exclusively in regions where goitre was endemic; (2) the malady was always limited to detached premises (particularly barracks, and, next to them, seminaries or boarding-houses), while no notable increase of goitre showed itself among the population of the place outside these institutions;⁵ (3) in the epidemics among the military, it has very often happened that only one barrack, or one section of the troops, has been affected, all the rest remaining exempt; and (4) the malady has affected mostly those of the troops who had just come to the particular garrison, chiefly the younger soldiers, almost exclusively the common soldiers, rarely the sergeants or corporals, very rarely and indeed quite exceptionally the officers.

¹ Hancke, in 'Hufeland's Journ. der Heilkde,' 1838, May, Bd. 86, Heft 5, 77.

² 'Reuss, 'Württemb. med. Correspondzbl.,' 1836, vi, 168.

³ v. Seidlitz, in 'Virchow's Arch.,' Bd. 86, 168.

⁴ L. c., 85.

⁵ The single exception to this is the small epidemic of 1833, in the Pauline Charity at Stuttgart, when several cases of goitre were observed simultaneously outside the institution, but in the same part of the town in which the latter was situated.

§ 42. INDEPENDENT OF CLIMATE, SEASON, OR WEATHER.

Of all the diseases that are met with in wide diffusion over the globe none appear to be, in their endemic occurrence, *so independent of geographical position or of climatic influences* as goitre and cretinism; while none, at the same time, are more intimately bound up with conditions of the soil. Both diseases occur with equal frequency in all latitudes, from the equator (Soudan, South America) to the arctic zone (Hudson's Bay Territory), in regions with a mean annual temperature of 85° Fahr. and upwards (Abyssinia, and other negro countries), and with one of 38° Fahr. and below it (Faluh in Sweden, Fort Kent in Maine, Irkutsk); and if many observers¹ have laid special stress on high degrees of atmospheric moisture as an essential condition of endemic goitre and cretinism, it has also to be taken into account that there are other regions counting among the chief seats of these maladies, such as Ladak, the province of Mendoza in the Argentine Republic, the Brazilian provinces of Minas Geraes and Goyaz, and Peru, where the atmosphere is an absolutely dry one.

In discussing the question of the dependence of goitre upon conditions of climate, on the basis of his experiences in the Andes of New Granada, Humboldt says: "Goitre is prevalent not only in the lower and the upper basins of the Rio Magdalena (*i.e.* from Neyva to the confluence of the Magdalena and Cauca), but also upon the mountain-ridges of Bogota, 6000 feet above the bed of the river. The first of those three regions is a dense forest, while the second and third consist of ground that is almost entirely barren; the first and the third are both alike moist, and the second is dry; and whereas the second and third are swept by the strongest winds, the atmosphere in the first is stagnant. Over the whole valley of the Magdalena the thermometer stands at 22° to 23° C. (71° to 73° Fahr.) throughout the year, while, on the plateau, it ranges between 4° and 17° C. (39° and 62° Fahr.); so that the climate of the one region is distinguished by its high and steady tem-

¹ Ackermann, p. 83, for Switzerland; Wenzel, p. 95, for the Salzburg Alps; Rösch, 'Untersuchungen,' p. 218, for Würtemberg; Vogt, for Lower Franconia; Marsden, for Sumatra, and others. Fodéré, who at one time entertained this belief (in 'Traité,' Germ. Transl., pp. 44, 140), subsequently held it to be erroneous. "At the present day," he says, "having had a larger experience, I no longer venture to assert that humidity is the only cause of cretinism and goitre" ('Traité de méd. légale').

perature, and of the other by its low temperature and great variations. It is precisely in the upper valley of the river (above Honda), where droughts and high winds prevail, that we find goitre to be much more common than on the banks of the river somewhat below Honda, where the inhabitants are continually subject to a damp and stagnant atmosphere." Restrepo had already written to the same effect concerning the seats of the disease in the Andes of South America. Subsequent experiences there led Boussingault and Foote to the same result, as the observations of Grange and Maffei in the Alps had done for them.

"Il n'est pas nécessaire," says Saint-Lager,¹ "d'aller jusqu'en Nouvelle-Grenade pour trouver des exemples analogues : quiconque a parcouru les Alpes et les Pyrénées, a vu des vallées voisines l'une de l'autre, ayant même direction, mêmes altitudes, même température et humidité, et présentant d'étonnants contrastes sous le rapport de la santé de leurs habitants." "With such facts as these," says Bramley,² referring to his observations in the Himalaya, "the reader will readily perceive the utter futility of the opinion that any one state of climate can be assigned as the universal cause of the disease . . . for the facts I have stated show, that it appears under all states and conditions of the atmosphere and every variety of climate."

A few observers, relying on their own observations, have laid special stress on the influence of season or weather on the development of the so-called epidemics of goitre; they have pointed out that these epidemics had occurred, or at least reached their height, uniformly in the summer season, and they have accordingly applied to such cases the name of "goître estival."³ But this is a view that also lacks confirmation when we come to sum up the various experiences; many epidemics of goitre have appeared and run their course at other seasons as well, and in fact it is impossible to show even a preponderance of the malady just at the time of high temperature.

Of 24 epidemics of goitre in France, for which I find the period of occurrence given with some accuracy, the respective seasons were as follows:

5 in winter	6 in summer
1 „ winter and spring	3 „ summer and autumn
7 „ spring	1 „ autumn
1 „ spring and summer	

The epidemic of goitre in 1877 among the Russian troops at Kokaun happened in the month of February. The epidemic of 1820 in Silber-

¹ 'Etudes,' p. 138.

² L. c., p. 224.

³ Nivet, 'Gaz. des hôpit.,' 1852.

berg originated in spring ; on the setting in of fine weather, the number of cases increased very slowly, but in the cold and wet autumn following, it rose so considerably that 100 new cases came under observation between the 17th and 20th November, and in December there were only 70 men, in a battalion 380 strong, who had escaped the disease.

§ 43. INFLUENCE OF LOCALITY.

All the more marked and unambiguous is the connexion that may be traced between endemic goitre and cretinism and the locality, or, in other words, between them and the *soil* or what the soil carries and contains. However widely the two diseases (and goitre in particular) are spread over the globe, and however large the tracts of country overrun by them, it is always and everywhere only a few spots, often narrowly circumscribed, which form the seat of the endemic ; outside these, even in the most immediate neighbourhood, the immunity is complete. The report of the Sardinian Commission brings out in the clearest terms this mode of incidence for the localities of Piedmont which are affected by goitre and cretinism ; and equally so the statements of Rösch for Würtemberg and of Maffei for Salzburg. The latter have been recently substantiated by Klebs, who points out that the disease cannot be dependent on atmospheric influences (sunlight, air, and the like), inasmuch as it is always limited to particular spots, the surrounding country remaining completely free from it. In the Hungarian countries of Pesth, Raab and Wieselburg, goitre and cretinism are found, according to Glatter, in the districts on the right bank of the Danube ; only an occasional case occurs on the left bank, while the more remote regions of even the right bank are free from both diseases. In the neighbourhood of Passau, there are only sporadic cases of goitre and cretinism, while in the Austrian village of Engelhardszell eighteen miles off, the cases amount to 10 per cent. of the population.¹ In Middle and Lower Franconia cretinism is met with at scattered points only.² In the quondam Duchy of Nassau it is only the town of Herborn and a few villages in the lofty Westerwald that form the endemic seats of goitre ; in all other parts of the

¹ Friedrich, 'Bayr. ärztl. Intelligenzbl.,' 1855, Nr. 28, 352.

² Majer, l. c.

country the disease is a rare phenomenon.¹ In the small section of the arrondissement of Rouen which is affected by endemic goitre, there are 49 villages situated close together, and of these only 25 are subject to the disease, the remaining 24 being completely free from it.² In Bedfordshire, goitre is endemic in a single village, Ridgemont, while all the country round is exempt.³

Incontrovertible proof of the influence of locality upon the production of goitre is furnished by the fact that healthy persons coming into goitrous spots from non-goitrous places not unfrequently contract the disease after a longer or shorter stay, and sometimes after a very short stay; secondly, by the fact that a change of locality has been found to be the most certain means of overcoming the disease or preventing its farther development; and thirdly, that in regions where goitre is endemic, the animals also are affected by it, especially the domestic animals such as dogs, cats, goats, sheep, pigs, horses and mules.⁴ As regards the first point, I may call attention to the fact already mentioned that, when goitre has broken out as an epidemic among the military, the troops to suffer have been chiefly those that have come into a goitrous locality from a non-goitrous. There are, besides, other references to persons previously healthy falling ill with it under the same circumstances, such as those by Erlenmeyer⁵ for the Rhine country, Glatter for Hungary, and Wilson for the Punjaub. Observations as to the good effect of a change of locality on the course of the disease have been published by Guyon⁶ from Santiago (Chili), Mendoza (Argentine Republic), and Switzerland, by Wother- spoon from Fort Kent, and by Greenhow from Oudh.⁷ Von

¹ v. Franque, l. c., 621, 623, 625, 627.
Blower, l. c.

² Vingtrinier, l. c.

⁴ This fact was known to Pliny, who says (l. c.): "Guttur homini tantum et suibus intumescit, aquarum quae potantur pleurumque vitio." Instances of the occurrence of goitre in animals are reported from all goitrous localities: by Keyssler, Fodéré and Saint-Lager (Piedmont), Baillarger (especially in mules) and Anzouy (Pyrenees), Moretin (Jura), Saint Lager (Lyonnais), Guerdan (Baden), Rösch (Württemberg), Mittermayer (Pinzgau), Gmelin (Lena Valley, Irkutsk), Bramley, Greenhow, McClelland, Campbell and Fayrer (India), Barton and Wotherspoon (United States).

⁵ 'Archiv,' l. c.

⁶ 'Gaz. méd. de Paris,' 1862, p. 345.

⁷ L. c., 441: "That change of locality is beneficial or the contrary to goitrous

Seidlitz informs us that when the number of cases among the Russian troops in Kokaun (Turkestan) reached such a height as to cause anxiety, the best results followed the removal of the whole military contingent to the neighbouring town of Margelan.

§ 44. INFLUENCE OF ALTITUDE AND CONFIGURATION.

The question now arises, What are those *conditions of soil*, common to all centres of goitre and cretinism, which can be brought into direct or indirect relation with the pathogenesis? A glance at the area of distribution of both diseases shows that their endemic occurrence is not by any means dependent as a universal rule upon a certain *elevation and configuration of the ground*; although it is true that they are mostly endemic in mountainous districts (most of all in high mountain ranges such as the Alps, the Himalaya and the Cordillera), while they are rare on table-lands, extremely rare on low levels, and never found hitherto on coast margins.¹

Saussure,² on the strength of his observations in the Swiss and Savoy Alps, was of opinion that the two diseases find their upper limit at a height of 1000 metres (3300 feet); while Demme and Maffei³ thought that they could make out a corresponding inferior limit at about 300 metres (1000 feet); so that, on either side of that zone, goitre and cretinism would occur only sporadically. But these fixed limits have a purely local value; for the general distribution of the disease, they cannot be applied at all. That the downward limit (reckoning to the sea-level) is a vanishing one, may be concluded from the prevalence of goitre in the northern plain of France (Departments of the Aisne and Oise), in various parts of England (Norfolk, Somerset and other

tumours, appears to be a proof that they depend, to some extent at least, on local causes. In England, in America, in Switzerland, and in India, it has been remarked that goitres decrease, and even disappear sometimes, on the patient's changing his residence."

¹ See Hutchinson, 'Med. Times and Gaz.,' 1855, Oct. This circumstance is most noticeable in those countries where goitre is widely prevalent in the plains situated at the foot of mountain slopes, although it leaves the proper coast region entirely free, as in Brazil (Rendu).

² L. c., ii, 487.

³ L. c., p. 147.

counties), and along the great lakes of North America (Michigan, Lower Canada); while the limit upwards which Saussure has fixed depends, as the Sardinian Commission¹ have pointed out and Maffei's data for the Salzburg Alps confirmed, upon the fact that the greater part of the cultivated land and the larger number of human habitations do not reach beyond the altitude named. As a matter of fact both diseases are found at still higher elevations: in the Hautes Alpes at 2060 metres or 6750 feet (St. Véran), in Savoy at 1566 metres (Albiez-le-Vieux, where there are 90 goitrous or cretinous persons in a population of 1000), in the Pyrenees at 1316 (Barèges), in Val Tellina at 1300 (according to Strambio), in Sondrio at 1700 (Livigno), even in Baden at 1000 (Hammereisenbach), in the Himalaya at 2000 (in Ladak and Nepaul up to 4000), and in the Cordillera of New Granada at from 3000 to 4000 metres (10,000 to 13,000 feet).

Goitre and cretinism, accordingly, cannot be shown to be dependent for their endemic prevalence upon elevation of the ground. And there is just as little truth in the other doctrine founded by Saussure,² and formerly in somewhat general currency, according to which deeply cleft valleys, receiving little sunshine or wind, and with a damp air or marshy soil, form by far the greatest if not the only seats of endemic goitre and cretinism. The goitrous and cretinous districts of the Piedmontese and Lombard Alps extend far into the plain of Upper Italy; in Switzerland we find both diseases on the low ground of Malters (Lucerne) which, as Troxler says,³ is neither narrower nor deeper than hundreds of other localities where there is no goitre or cretinism, and, again, in the wide and exposed valley of the Aar; while the occurrence of cretinism at

¹ L. c., p. 173.

² L. c., ii, 390, 480. Saussure's contention is that, whereas the inhabitants of the upper part of a valley appeared to be perfectly healthy, he began to find the first traces of cretinism when he came to hamlets in deeper situations, the number of cases rising proportionately as he descended towards the bottom of the valley. Beyond the point where the valley began to open on to the level, the malady became rarer, and on the plain itself, as well as in the open and wind-swept valleys, it vanished altogether; although the conditions of living, as he adds, were exactly the same for the whole population of the valley.

³ L. c., p. 43.

Langenargen on the Lake of Constance furnishes us with a classical instance of the disease having its seat in an open plain. In the Lyonnais goitre occurs equally on the plain and in the valleys, in Alsace and the Palatinate it is prevalent on the level expanse of the Rhine valley, in Lower and Middle Franconia we find it (along with cretinism) not in valleys but on the slopes of the Steigerwald, in Thuringia it is in mountainous and in level districts equally (according to Rehm and Kirchhoff), in Upper and Lower Austria on the great plain of the Danube, in Salzburg on the low grounds of the Enns and Traun, and in Styria on the broad plain of the Mur (the Eichfeld).¹ One of the most important goitrous regions of India, as we have seen, is the plain of the Terai joining on to the slopes of the Himalaya. In Northern China, the endemic of goitre is by no means confined to the mountainous regions, but extends over the level country as well;² and the same is true of the western parts of the Soudan.³ On the western slope of the Cordillera of Mexico, the focus of disease goes as far down as the bottom of the basin;⁴ and in like manner, goitre extends from the eastern slopes of these mountains far into the plains of the southern provinces of Brazil and into those of the Argentine Republic.

§ 45. RELATION TO THE GEOLOGY AND MINERALOGY OF THE LOCALITY.

A special interest attaches to the question whether any connexion can be made out between the endemic occurrence

¹ Köstl, who lays most stress for the pathogenesis upon the valleys being shut in, finds examples of this in the valley of the Mur. Between Predlitz and Murau, where the valley is 400 to 500 paces wide, there is one cretin to about fifteen inhabitants; from Murau to Schleifing, where the valley becomes four times as wide, cretinism becomes three times less common; from Schleifing onwards, as it narrows again, the malady almost doubles; and as the valley widens somewhat from Unzmark to Judenburg, the disease once more becomes less frequent. So far so good. But now the valley opens out from Judenburg down to the level of Eichfeld, where one would expect cretinism to vanish; but here, we are told, the conditions favorable to cretinism are so strongly developed that the number of cases at certain spots is from 5 to 10 per cent. of the whole population.

² Dudgeon, l. c.: "It is found also on the plains and in our large cities."

³ Quintin, l. c.

⁴ Matthieu de Fossey.

of goitre and cretinism and the *geological or mineralogical character of the soil*. This is a question which has been discussed with more or less of keenness by nearly all the more recent observers, although as yet they have come to no conclusion that is at all satisfactory. The difficulty attending the solution of the problem lies essentially in the fact that there is a want of exhaustive and thorough information as to the conditions of soil in the individual circumscribed localities which are the seats of goitre and cretinism; as well as in the fact that, in forming our opinion, we should require to know not merely the upper layer of soil but also the character of the bottom, which is often very different from the surface; and, further, in the fact which has been quite rightly dwelt upon by Schwalbe,¹ that some localities contain minerals either in the form of ore-deposits or of lodes, or to the extent of a mere impregnation, and that these impart to the soil a geological character which may escape observation for a long time. While paying the fullest tribute to the thoroughness of the inquiries directed upon this question by McClelland,² Billiet,³ Grange,⁴ Saint Lager, Garrigou,⁵ and others, we can hardly regard the results that they have arrived at as conclusive; for, apart from the discrepancies among themselves, they cannot always be brought into harmony with the conclusions of observers in other parts of the world; and as the latter have often been superficial in their determination of the geological conditions, we are without the means of deciding critically wherein the contradictions and discrepancies lie, and how they are to be reconciled. I think it well to introduce the following table with that remark, so that the reader may have a correct appreciation of the trustworthiness of the conclusions drawn from it.

¹ 'Correspondbl. des Thüring. ärztl. Vereins.'

² 'Sketch of the Medical Topography, or Climate and Soils of Bengal and the N. W. Provinces,' Lond., 1859.

³ 'Annal. méd.-psychol.,' 1854, April, 1855, Jan.

⁴ 'Gaz. méd. de Paris,' 1848, 820, 1849, 972, 1850, 554, 1851, 103; 'Arch. gén. de méd.,' 1850, Jan., 108, Oct., 243.

⁵ 'Bull. de l'Acad. de méd.,' 1868, xxxiii, 915; 'Gaz. hebdomad. de méd.,' 1874, 270, 284.

Tabular Survey of Endemic Goitre and Cretinism as occurring on the several Geological Formations.¹

Geological Formation.	A star (*) before the name of a locality denotes both goitre and cretinism as endemic; places not so designated have goitre only.
Oldest and Eruptive Rocks (Gneiss, Mica-Slate and Clay-Slate, Granite, Syenite)	*Piedmont (valley of Aosta, Tarentaise, Upper Savoy)—*Val Tellina (valley of the Adda)—*Norican Alps (Upper and Lower Austria, Styria)—*Switzerland (Bernese Oberland, Vallais, Grisons)—*Transylvania (circle of Kronstadt)—*Sudetic Mountains—*Erzgebirge (Annaberg and other places)—*Harz (Lautenthal)—*Baden (Neustadt)—Nassau—Sweden (Faluh)—Finland—Alabama—New Granada (Pamplona, Socorro).
Silurian and Devonian (transition, Grey-wacke)	*Norican Alps (Salzburg, Tyrol, Styria)—*Pyrenees—Vosges—Sudetic Mountains—Harz (Lerbach, Clausthal)—Siberia (basin of the Lena)—*Himalaya (Kumaon, &c.)—Hudson's Bay Territory (shores of the Elk and Peace Rivers)—New Granada (on the mountain ridge between Villata and Muzo)—Brazil (Prov. of Goyaz, especially the western slopes of the Sierra Geral).
Coal . . .	England (Derby, Nottingham, Yorkshire, Cumberland)—Silesia—Pennsylvania (Pittsburg, &c.).
Permian . . .	*Val Tellina (valley of the Lire)—*Maritime Alps—Lyonnais (Rhône)—Hesse (especially valley of Neckar)—Thuringia—New Granada (eastern slopes of the Cordillera).
Trias. { Variegated Sandstone	*Norican Alps (Styria, Tyrol)—*Black Forest (eastern division)—*Lower Franconia (slopes of the Spessart, valley of the Main)—*Thuringia (Schmalkalden, &c.)—India (plain of Hindostan (Terai) on the slope of the Himalaya)—New Granada (valleys of Suarez, Chicamocha, Surata, &c.)—Peru—Chili.
Limestone	*Wurtemberg (region between Rottweil and Mergentheim)—*Sigmaringen (in a side valley of the Neckar)—*Baden (Neudenau, in the Jaxthal)—Hesse—*Lower Franconia (Erlabrunn, valley of the Main)—Thuringia—New Granada (province of Socorro).
Keuper	*Savoy (valley of the Isère)—*Hautes-Alpes—*Basses-Alpes—*Wurtemberg (chief seat of the disease in the circle of the Neckar and Jaxt)—*Lower Franconia (western slope of the Steigerwald, Sulzheim, Geroldshofen, &c.).

¹ This table, it need hardly be said, makes no claim to completeness; I have, for the most part limited it to the larger centres of disease.

Geological Formation.		A star (*) before the name of a locality denotes both goitre and cretinism as endemic; places not so designated have goitre only.
Jura.	Lias	*Piedmont (valleys of the Stura, Varaita, &c.) —Switzerland (Aigle, Ormonds, and other places in the Canton Vaud)—France (various localities)—Yorkshire (at a few places).
	Jura	*Norican Alps — *Savoy — *Dauphiné — *Hautes-Alpes—Department of the Meurthe (Oolite formation)—Yorkshire (Upper and Middle Oolite).
Chalk . . .		Seine-Inférieure—England (Norfolk, Buckinghamshire, Hampshire).
Tertiary . . .		*Switzerland (Bern, on the nagelfluh, several localities in the Canton St. Gall)—*France (Dauphiné, Basses-Alpes, &c.) — Lower Austria — Baden—*Wurtemberg (Langensargen).
Sedimentary . .		Italy (plain of the Po in Lombardy)—France (Bresse)—*Plain of the Rhine (Alsace, Palatinate)—Hungary (banks of the Danube and Drave, in the County of Baranya)—Argentine Republic (eastern basin).
Volcanic (trap) .		*Piedmont (a few places in Asti and Acqui)—*France (Upper Auvergne)—India (Chota, Nagpore, &c., probably on basalt)—Java—Mexico (Colima)—Azores.

Although this survey is far from an exhaustive one, it serves to furnish evidence of several facts. The first of these, already pointed out by Boussingault, by the Sardinian Commission, by Lebert, Grange, Nièpce, and others, is that no geological formation precludes the occurrence of goitre and cretinism. The second is that the two diseases occur much more frequently (although not exclusively, as Eschricht¹ thinks) on the older formations (including the Trias) than on the newer. The third is that they occur only on those sedimentary formations which are composed of the detritus of older rocks, as, for example, in the plains of the Rhine and of Lombardy, and in the valleys of the Arve and Doria.² “Dans la province de Savoye-Propre,” says the Sardinian Commission with reference to the last-mentioned fact,³ “le crétinisme endémique se rencontre seulement sur la rive

¹ ‘Verhandl. der Würzb. phys.-med. Gesellschaft,’ 1854, iv, 141.

² See Saint-Lager, ‘Études,’ 443.

³ P. 67.

gauche de l'Isère, qui fait suite aux Millières, de la province de Haute-Savoie. Les crétins cessent aussitôt, qu'on arrive sur les terrains de calcaire crétacé ou jurassique. Monseigneur Billiet, archevêque de Chambéry, dans les renseignements par lui fournis à la commission, assura que parmi les 140 paroisses situées dans ce dernier terrain, 7 seulement présentent des traces de crétinisme; encore ces communes se trouvent-elles sur un terrain tertiaire formé de detritus des Alpes et sur une molasse argileuse."

Garbiglietti and Ferraris make similar statements for other parts of Piedmont. Rösch, after sketching the distribution of goitre and cretinism in Würtemberg on the variegated sandstone, muschelkalk, and keuper, goes on to say: "Between the keuper and the Jurassic limestone, forming the main chain of the mountain, there is a narrow zone of Liassic limestone and Liassic slate at the foot of the Alp, also running from south-west to north-east. On that, as well as on the whole Jurassic formation, goitre and cretinism are nowhere endemic,"—a fact which had been pointed out before by Riedle as regards goitre, when he proved from the recruiting lists that, in 1000 conscripts examined, there were 130 to 155 goitrous among those from the communes on the Trias, but only 2 to 3 per 1000 from localities on the Alp.

After what has been pointed out already as to local delimitations of the foci of disease, often within very narrow bounds, and as to restriction to particular villages (the adjoining localities being entirely exempt) and as to the immunity of certain points in the midst of a large area of the disease although the geological formation is everywhere absolutely the same, it hardly needs more evidence to show that the geological character of the formation *in and by itself* can by no means determine the occurrence of goitre and cretinism. It is remarked by Boussingault that in the mountain ridges which run along the coast of Venezuela, one encounters granite, gneiss, mica-slate, talc or clay-slate, and that the same formations enter into the soil of the plain of Caracas and the valleys of the Aragua and Tuy. Now, whereas goitre is very rare throughout the whole province of Caracas, the malady occurs in general diffusion in the provinces of Pamplona,

Bucaramonga, Giron and others. Throughout a great part of New Granada we find syenite and porphyraceous green-sand in such localities as Montuosa Baxa, Cacota de Balesco, and Laxas, where goitre is endemic; whereas in the province of Antioquia and over a large part of the upper valley of the Cauca, which belong to the same formation (oldest eruptive rock), the disease does not occur. Boussingault found many villages much affected by goitre (Villata, La Palma, Copes el Peñon, Pacho, and others) on a belt of clay-slate running northwards from Villata towards Muzo; whereas, on a second belt of the same formation in the Eastern Cordillera, forming the water-shed between the valley of the Magdalena and that of the Cauca, not a single case of goitre is to be met with. There are the same discrepancies in the distribution of goitre in New Granada on the red sandstone, the bunt-sandstone, and other formations.

§ 46. INFLUENCE OF WET SOIL.

If, then, there be some causal connexion between the endemic occurrence of goitre and cretinism and the nature of the soil, and that connexion do not depend on the geological formation, then the determining factor must lie either in the *physical characters of the soil* or in its *chemical constitution or mineralogical characters*.

In regard to the former of these, many observers have laid special stress upon the *copious saturation or swamping of the soil*; and it has been specially remarked by some¹ that, where goitre and cretinism occur upon the oldest eruptive formations (the Primary rocks) or upon the older formations generally, the explanation is partly in the fact that the kind of cleavage of the ground peculiar to these formations leads to the production of deeply-cleft, winding, and therefore very damp valleys, such as have been shown by experience to be the chief seats of the disease. It will appear from what has been said of the occurrence of the disease in wide and open valleys and on plains, that this latter argument carries only a certain amount of weight. But those other facts which would

¹ Especially Garbiglietti, 'Giorn. delle sc. med. di Torino,' 1845, Giugno.

make the distribution of the disease to depend upon dampness of the ground in general have not the significance that has been often ascribed to them, inasmuch as there are just as many and just as trustworthy observations going to show that goitre and cretinism are indigenous and fully developed even on the driest of soils.

In the Cordilleras of New Granada Humboldt found that both diseases were as common in valleys with a dry soil as with a wet, and that certain wooded districts characterised by heat and moisture, such as the province of Antioquia, the banks of the Orinoco, Cassaquiri, and Rio Negro enjoyed complete immunity from the disease. Roulin and Boussingault have expressed a similar opinion, the former stating emphatically¹ that among the Cordilleras goitre is prevalent in districts which lie quite exposed and count among the driest spots on the globe ("qui sont les plus sèches du monde"). Referring to the distribution of goitre in North America, Barton² points out that the malady has its seat principally in damp valleys and on swampy river banks; on the other hand, Denny observes that Pittsburg, which is affected by endemic goitre, stands upon a perfectly dry plateau, the vicinity of the town being quite free from marsh. One of the most considerable centres of goitre and cretinism in the Rhone valley is the district of Aigle, notwithstanding that it is free from marsh, moderately dry, exposed to the sun all the year, and well swept by winds.³ "In the Alpine valleys," says Troxler,⁴ speaking generally, "and in other valleys where cretinism occurs, there are almost no proper marshes but only occasional collections of water, producing no important consequences; on the other hand, in the marshiest of regions. . . . cretinous lesions have not been discovered even by the most careful observers." Wenzel,⁵ Streinz, Hofer, Ozlberger, and various others who have discussed the question with reference to the diseases as they occur in the Norican Alps, lay special stress upon the dampness of the soil in the goitrous centres. Maffei also takes that factor to be not irrelevant, adding, however: "but I know very well that even the suniest, brightest, and driest situation may not preclude the development of cretinism;" and in order to show how little a marshy soil can determine the occurrence of the disease, he points to the marshes and moors of low countries, and to the shores of lakes and great rivers, which are quite exempt from goitre and cretinism. Schaussberger, who treats of the endemic goitre of Upper and Lower Austria, observes that while many villages, such as Seissenheim, Krum-Nussbaum, and Gross-Pöchlarn are swarming with cretinous and goitrous persons, there are other villages in their

¹ 'Gaz. méd. de Paris,' 1845, 690.

² L. c., 91.

³ Lebert, 'Archiv,' l. c.

⁴ 'Schweiz. Archiv der Med.,' 1817, Heft 3, 49.

⁵ L. c., p. 96.

⁶ L. c., p. 154.

immediate neighbourhood and with the same low and damp situation which are affected but little or not at all, such as Aschach, Ottensheim, Ybbs, and particularly Marbach, which is only some ten minutes' distance from Krum-Nussbaum, and is in all other respects similarly placed with it. The same discrepancies come out on comparing the conditions of soil in those localities of Würtemberg which are subject to goitre and cretinism. While Kerner, Dürr, Rampold,¹ and others, emphasise the dampness of the soil as an essential part of the causation, it is observed by Rösch,² who agrees with them generally, that "the valleys of the Alb, which are often very narrow and deep, especially the Donauthal and the Brenzthal, are damp, full of mists and marshy in places, as, for example, the fine stretch of the Donauthal from Mühlheim through Tuttlingen to Sigmaringen, and yet goitre is very rare there and the cretinous degeneration does not occur at all." Faber's opinion is to the same effect.³ The centres of goitre and cretinism in Middle Franconia (Iphofen, Einersheim, &c.) lie high and exposed on a dry plain bounded by the Steigerwald.⁴ While special stress is laid by Tourdes,⁵ Herrmann,⁶ and others on the marshy character of the Rhine valley as a cause of its endemic goitre and cretinism, Müller⁷ on the other hand has shown by his observations that the valley of the Neckar in Hesse, which is also a seat of goitre and cretinism, is distinguished by the absolute dryness of its soil. Again, Lettsom would regard the wetness of the soil in Derbyshire as helping materially to produce the endemic of goitre there; while we have to note, on the other hand, from Rumsey's observations on goitre around Beaconsfield (Bucks), that the town itself is free from the malady although its situation is more damp than the adjoining valleys which are affected with goitre.

It cannot be denied that a wet or marshy soil is anything but a matter of indifference to the well-being of the residents upon it, or that the state of health does experience a change for the better when that evil is overcome, or, in other words, when the soil is dried by drainage, the making of regular water-courses, and the like. And we are indeed justified in associating with that hygienic progress the decrease of goitre and cretinism which has been experienced in the valleys of Savoy,⁸ in the Pyrenees,⁹ in the valley of the Rhine in Alsace,¹⁰ in the Jaxtthal in Würtemberg,¹¹ and

¹ 'Württemberg. med. Correspondenzbl.,' 1835, v. 159.

² L. c., 218.

³ L. c., 221.

⁴ Hoffmann, 'Einiges über den Cretinismus u. s. w.,' Würzb., 1841.

⁵ L. c., 53.

⁶ 'Blätter für gerichtl. Med.,' 1882, 147.

⁷ 'Bad. med. Annal.,' 1839, v. 89.

⁸ Fodéré, p. 190; 'Report of the Sardin. Commission,' p. 200; Dubini, l. c.

⁹ Boulinière, l. c.

¹⁰ Tourdes, Herrmann, ll. cc.

¹¹ Kerner, l. c.

at other places where such improvements in the soil have been carried out. But it is clear that we are here concerned, not with obviating a specific factor of disease, but with a general elevation of the state of health of the residents and with their increased power to resist morbid influences.

§ 47. CONNEXION WITH LIMESTONE, AND WITH MAGNESIA IN PARTICULAR.

According to the oldest and most general view of the origin of goitre, accepted by most observers down to those of the present day, the cause of the disease is to be found in the habitual use of water rich in certain mineral constituents. And, inasmuch as the presence of mineral substances in the water depends on the ground from which it springs or over which it flows, and as, by experience, the suspected "goitre-springs" are found to be particularly those with carbonate of lime or gypsum dissolved in somewhat large quantities, it was natural to conjecture that goitre and cretinism must be associated as endemics with a limestone soil—a conjecture which the accurate examination of soils at an early period of the inquiry tended to strengthen.

Boussingault was perhaps the first to call attention to this significance of a limestone soil—in the Cordilleras of New Granada. Then came Sensburg, Hoffmann, and Stahl, who pointed out the association of the two diseases with gypsum, marl, and other limestone-bearing soils in Lower Franconia. Riedle made out that goitre and cretinism in Würtemberg were found mostly on the muschelkalk and keuper, and next in order on the Jurassic limestone and molasse—a fact which was subsequently confirmed by Rampold, by Heyfelder for a side-valley of the Neckar in Sigmaringen, by Dürr for the circle of the Jaxt, and by Rösch for the circle of the Black Forest. Falck showed that in Hesse both diseases occurred mostly on muschelkalk, stratified limestone and zechstein, whereas the localities with lias, oolite, basalt, and clay were exempt from them; thus of 93 villages in which goitre and cretinism were endemic, 84 were upon zechstein and muschelkalk, 3 on primitive rock, 3 on clay, 2 on molasse, and 1 on trap. The same results were come to by Guerdan for Neudenu (Baden), both diseases being prevalent on muschelkalk; and by Maffei, in part at least, with reference to the distribution of these maladies in the Norican Alps.

The first thorough inquiry into the circumstances in ques-

tion was instituted by McClelland in the province of Kumaon on the slope of the Himalaya. His results were as follows :

In 91 villages situated on granite and gneiss, hornblende slate and mica slate, clay slate, green sandstone, granitine, and silicious sandstone, having an aggregate population of 5383, there were 29 goitrous persons and no cretins; whereas in 35 villages on Alpine limestone (*i.e.* Jurassic limestone and zechstein), having an aggregate population of 1160, 390 cases of goitre were found and 34 of cretinism.

These results were afterwards confirmed by Thorel for Mekong and Cochin China. "A mesure que les montagnes de calcaire deviennent plus nombreuses," he says, "les cas de goître sont également d'une extrême fréquence. Il suffit, pour que le nombre des goitreux augmente, qu'il y ait près des villes et des villages, des montagnes de marbre."

In the meantime Billiet¹ had been making inquiries in the same manner as McClelland, into the relation between goitre and cretinism and the various kinds of soil in the Diocese of Chambéry (Savoy); and he satisfied himself that, of 169 villages, 127 were free from the diseases in question, while 42 formed endemic seats of the same. Further, he made out that the cases were occasional on the alluvium of the Rhone and on older diluvial formations, that the number of cases rose on coming nearer to the argillaceous limestone soil which runs from Montmélian to Chamousset, and that the endemic reached its height on the limestone, magnesia, and gypsum of the valley of the Maurienne, where the cases amounted to 10 per cent. of the population. While the whole of the 127 villages on Jurassic and neocomian rock enjoy an absolute immunity, the disease begins to appear in endemic form on argillaceous limestone and slate, and most of all upon soil which bears talc-slate, micaceous slate and gypsum. That it is here the state of soil only that is the determining factor is inferred by Billiet from the fact that all the valleys in the diocese, both those subject to the diseases and those exempt from them, are absolutely alike in all other

¹ L. c., 172.

² 'Mém. acad. de Savoye,' 1847; 'Annal. méd.-psychol.,' 1854, April, 185 Jan.

circumstances—as regards the form of the valleys, their steepness, their planting, their exposure to the sun, their hydrology, and their buildings, and as regards the well-being of their inhabitants.

While each of these two authorities had directed his inquiries exclusively upon one small area, Grange¹ extended his investigations on the subject over a wide territory, studying the problem in the Pyrenees, the Vosges, and in the Alps of Piedmont and Switzerland. He came to the conclusion (which Zambroni² had come to long before him) that it was in no way an affair of the whole mass of limestone rock, but exclusively of the magnesia in it, the maximum amount of the disease being found on a soil of dolomite or magnesian limestone.

Grange's first observations were made in the valley of the Isère; both there and in the diluvial plain of Grenoble, in the valleys of the Vosges, Jura, and Pyrenees abounding in dolomite, on the molasse (nagelfluh) of Western Switzerland, in the departments of the Oise, Aisne, and Somme, in a few districts of Dauphiné, and in the departments of Haut-Rhin and Bas-Rhin, it was the abundance of magnesia in the soil that appeared to determine the occurrence of goitre and cretinism. However various the elevation, the configuration and the formations of these regions might be, one unvarying factor in them all was the presence of magnesia in the rock, whether it occurred in the form of magnesia-containing silicates (as particularly in gneiss and granite and in hornblende-rocks) or in the form of dolomite; and it was the absence, or the somewhat scanty or infrequent occurrence of magnesia in the younger Jurassic rocks, in the chalk, and in the Tertiary formations that explained the immunity of localities in whose soil these predominated.

These conclusions of Grange on the relations of a particular quality of soil to endemic goitre and cretinism are in agreement, not only with earlier observations as to the prevalence of both diseases on the Trias (muschelkalk, keuper, and zechstein), and on the transition limestone of Lower and Middle Franconia, Würtemberg, Sigmaringen, Baden, the Norican Alps, and India; but they have been confirmed also

¹ 'Compt. rend.,' 1848, ii, 358; 1849, ii, 695; 1850, i, 518, ii, 58; 'Annal. de chimie et de phys.,' xxiv, 364; 'Arch. gén. de med.,' 1850, Jan., 108.

² In the paper brought out by Mongez ('Revue méd.,' 1825, iv, 139), on the occasion of the discussion at the Académie des Sciences upon the memoir drawn up by Roulin relating to the distribution of goitre in New Granada.

by many recent observations at the most diverse points within the distribution-area of the two diseases—by Morel¹ and Ancelon for the Vosges, by Allaire and Richon for the vicinity of Diedenhoffen and Metz, by Gaudin for the department of Nièvre, by Anzouy and Garrigou for the Pyrenees, by Virchow and Vogt for Lower Franconia, by Majer and Rüdél for Middle Franconia, by Röhrig for the village of Aurach (Waldeck), by Gray for Bhootan, by Greenhow for Oudh, and by Tschudi for Brazil (especially the province of Minas Geraes from Ouro Preto upwards, with its “itacolumit” or magnesian-quartz soil).

Lower Franconia, as Virchow points out, belongs to the Trias, excepting its northern and western part; the larger part of the Spessart rests on variegated sandstone, the declivities of the valley of the Main yield muschelkalk, and the Steigerwald, a range extending in all directions throughout the Franconian Highlands, consists of keuper. Goitre and cretinism in this region are endemic upon muschelkalk; on the other hand, both diseases are entirely absent from the interior of the Spessart where the variegated sandstone is most abundant, beginning to show endemically at the edges of that formation, where muschelkalk overlies the sandstone, as well as on a belt of it abounding in salt springs. At the highest points of the Steigerwald, the upper members of the keuper predominate (sandstone mixed with argillaceous strata); at the foot of the range and in the plain, extensive layers of dolomite are exposed, among which gypsum crops out. Thus the area of disease is here limited to localities where muschelkalk, dolomite or gypsum in keuper-marl are found, or where strata of limestone and zechstein occur side by side with variegated sandstone.²

The theory developed by Grange and supported, as we have seen, on many sides, has not been permitted to go unchallenged. Thus it has been contested by Saint Lager, whose very comprehensive study of the geological characters of soils over the whole globe (so far as they are known) has led him to the opinion that goitre and cretinism are indigenous only in regions with metal-yielding rock, that their endemic occurrence depends essentially upon the presence of sulphuret of iron or of copper pyrites, and that

¹ In ‘Congrès scient.,’ Nancy, 1851; ‘Annal. med.-psychol.,’ 1854, avril; ‘Traité des dégénérationes phys., &c.,’ Par., 1857; ‘Arch. gén. de méd.,’ 1864, fevr., 173; 1865, juill, 5.

² I may note that dolomitic rock is predominant also in the Western Soudan, where Quintin has found goitre to be endemic.

their prevalence on soils containing magnesia is explained by the fact that that rock is especially liable to contain sulphuret of iron. This view has lately found support in the inquiries of Lebour on the distribution of goitre in England.¹ But against it the objection has been raised by Garrigou that there is not a trace of endemic goitre in those very districts of France where sulphuret of iron occurs in largest quantity—in the canton of Ax (Dept. Arriège) and in the canton of Alais (Dept. Gard); while the disease is endemic in many parts of the country where not a trace of sulphuret of iron (or other metal) can be discovered in the soil. There is still another circumstance that I must call attention to as being not without significance for the question in hand: I mean the fact commented on by Thomson and others that goitre is wanting in New Zealand notwithstanding that in the northern island, in which almost the whole native population reside, large masses of magnesian limestone lie exposed.

Thus the results of inquiries instituted to discover the connexion between goitre and cretinism and the mineralogical character of the soil, do not justify us in coming to a definite conclusion. At the same time, the theory developed by Grange is noteworthy in the highest degree. But it still remains not altogether clear what is precisely the connexion between quality of soil and the development of the disease; at all events the doctrine deduced from these facts of some goitre-producing property in magnesian drinking-water is, as we shall see, a highly unlikely one.

§ 48. DIFFERENCES OF LIABILITY ACCORDING TO RACE.

With regard to the liability of various *races and nationalities* to goitre and cretinism, the statements of the several authorities are very conflicting.

Thus Greenhow and Barton state that they had not seen a single case of goitre among the white residents; whereas, according to the experiences of Wilson and Fayrer, the disease is equally common among all races. For Nicaragua, the accounts of Bernhard and Guzman agree

¹ 'Med. Times and Gaz.,' 1881, Oct., 492.

that goitre is most frequent among the Indians and more rarely met with among those of mixed blood. In New Granada it would appear, from Humboldt and Roulin, that the disease seldom occurs among the Indians, but, as Roulin adds, it is common among the negroes and the whites. In like manner Smith found goitre most frequent in Peru among the whites and the negroes, but rare among the mestizzos and Indians; while Tschudi found it in Brazil equally common in negroes, mulattoes, and whites.

Whether these differences, such as they are, in the amount of the disease in a mixed population, are attributable to racial distinctions, appears very doubtful; at all events, the above facts, as well as the fact that goitre is endemic among the Indians of North America (Barton), among the Malay population of Sumatra, Java, and Ceylon, among the Mongols in Ladak and China, and among the Arabs in Algiers and Morocco, make it undoubted that no race or nationality enjoys an immunity from goitre. But it is worthy of the fullest attention that, although goitre is widely spread in the Western Hemisphere, cretinism occurs much more rarely there than it does in the Eastern, or than it does on the soil of Europe in particular.

§ 49. NEGLECT OF HYGIENE A PREDISPOSING CAUSE.

Few diseases would seem to offer so favorable an opportunity for etiological research as goitre and cretinism. The delimitation of the morbid areas, for the most part within narrow bounds, might make it appear an easy matter to arrive at a knowledge of the physical or chemical influences peculiar to those particular localities as contrasted with others adjoining them,—influences such as could be brought into direct relation with the production of the disease or could be designated as its proper cause. And, indeed, the first observers thought that they had speedily and conclusively solved the problem. But the farther the inquiry proceeded and the larger became the number of places at which observations were made, the more marked were the differences of opinion that showed themselves among the observers, and the greater was the accumulation of diverse theories. An impartial

estimate at the present day requires us to admit that the *cause of endemic goitre and cretinism* is still enveloped in obscurity. I think I may be excused from recounting all the opinions that have passed current on this subject; and in the sequel I shall confine myself to a summary discussion of such theories as continue to the present day to have some importance assigned to them, or have been recently the subject of discussion.

The earliest observers were chiefly impressed with the fact that the centres of goitre and cretinism were to be found in deeply cleft, narrow, and damp valleys, little open to the sun or little swept by the wind, and they concluded that the essential cause of the disease was furnished by that kind of valley-formation, and by the high degree of atmospheric moisture, associated with high temperature and deficient sunlight, dependent thereon. This view was first put forward by Ackermann,¹ Fodéré,² and the brothers Wenzel,³ and it found defenders subsequently, although in a less exclusive fashion, in Berchtold-Beaupré (for Freiburg in Switzerland), Guista (for the Val d'Aosta), Pilz (for the Ennsthal), and others. When speaking of the relations that might be traced between these diseases and states of atmosphere and soil, I showed the untenability of this doctrine (subsequently abandoned by Fodéré himself), although I did not question the injurious influence on the health of the population of these factors in general, or their importance as predisposing causes. It is in the same sense, it seems to me, that we have to judge of a second class of etiological factors, to which a preponderant importance, especially for cretinism, has been attached by certain observers, viz. the noxious effects due to *social misery, insufficient food, drunkenness, filth, overcrowded and ill-ventilated dwellings, and neglect of body and mind*. The good results which have followed improved conditions of living among the inhabitants of particular places in Switzerland, Piedmont, Germany, and France (Vosges, Puy-de-Dôme) in limiting the extent and severity of cretinism, do not permit us to doubt that there are in social defects, just as in the already mentioned atmospheric and telluric influences, certain factors favorable to the production of the disease,

¹ L. c., 83.² L. c., 44, 140.³ L. c., 95.

the obviating of which is one of the most important and most profitable of hygienic tasks.¹ But there are certain facts which make it clear that we are not therein concerned with a specific cause of disease. These are :—that many localities which are quite exempt from goitre and cretinism present the same social conditions among their inhabitants as the places afflicted by those diseases, or even social conditions that are worse ; that the populous centres of Europe, Asia and America, where all those noxious influences are in the aggregate developed most, are precisely the places where goitre and cretinism do not occur, or occur in sporadic cases only ; that in many of the tracts of country afflicted with these diseases, the inhabitants are in the enjoyment of comparative comfort, goitre and cretinism where they are endemic being by no means exclusively the attributes of poverty and misery.

“ Je ne crois pas nécessaire,” says Boussingault, “ de réfuter l’opinion qui attribue le goître à l’ivrognerie, à la malpropreté, à l’usage d’alimens grossiers ; sans doute que les auteurs d’une semblable opinion n’avaient pas eu l’occasion de séjourner dans un pays où le goître est commun ; autrement ils auraient pu observer cette maladie chez les individus les plus sobres et dans la classe aisée de la société.” To the same purport is the statement of Saint-Lager with reference to the occurrence of cretinism even amidst comfortable circumstances : “ J’ai été fort surpris, après avoir lu tout ce qu’on a écrit au sujet de l’influence qu’exercent sur la production du crétinisme la misère, la saleté et la mauvaise nourriture, de trouver en Suisse, en Savoie, en Dauphiné et en Piémont des crétins dans les villes et les villages les mieux bâtis, dans les demeures les plus propres et chez les particuliers les plus aisés. Il est bien entendu, que je parle ici de l’aisance réelle et non de la richesse, qui n’empêche pas certains individus de vivre à la façon des misérables.” After quoting a large number of observations which he had made on this point, he goes on to say : “ La noblesse de Sion, de Sierre et d’Aoste a eu des crétins : ne pouvant citer des noms, par égard pour les familles, je me borne à affirmer que j’ai vu des crétins au sein des familles jouissant de la plus grande aisance.” It had been remarked previously by Fodéré that cretinism occurred “ equally in palaces and

¹ “ La miseria non è direttamente causa di cretinismo, ma ne è un elemento favoritore, incubatore ”—says Lombroso (l. c., p. 14) ; and Herrmann (l. c., 153) sums up his experiences of the influence of an injurious hygiene in producing the cretinism of the plain of the Rhine in Hesse, with these words : “ I am convinced that the social and domestic defects which have been mentioned are to be taken merely as unfavorable or complicating phenomena and by no means as causes operating independently for the production of cretinism.”

² ‘ Etudes,’ p. 173.

³ L. c., 72.

in thatched cabins;" and we have the same kind of testimony—in the writings of Troxler and Lebert for Switzerland, of Hoffmann for Lower Franconia, of Rüdel for Middle Franconia, and of Hermann for the Hessian plain of the Rhine,—that the malady is indigenous in localities where the fertility of the soil, the earning power of the inhabitants, and the comforts of living compare favorably with those of neighbouring communes which are quite exempt from the disease. That goitre is independent of these etiological factors is testified to not only by Boussingault, but also by Grange, Tourdes (for Alsace), Vingtrinier (for the department of the Seine inférieure), Berkowski (for Perm), and others. The opinion also that the epidemic outbreaks of goitre in French garrisons were specially connected with hygienic defects (overcrowding and filth in the barracks, unsuitable food, &c.)—a view adopted by several French military surgeons—has found no support in the experiences of Gouget, Viry and Richard, Fleury and Saillart. In order to dispose of this theory as absolutely untenable it is only necessary to add that the disease has been observed to occur in animals.¹

§ 50. THEORY OF INCREASED VASCULAR PRESSURE WITHIN THE THYROID.

Another theory, which applies especially to goitre, sets out with the assumption that, in the development of that disease we are concerned exclusively with a *hyperæmia of the thyroid gland, produced in a purely mechanical way, and continuing for a long period or recurring from time to time*, and that the occasion of it is furnished, sometimes by *pressure in the cervical vessels or in the thyroid directly* (as a result of long-continued extension or strain of the neck in certain attitudes of body), and sometimes in *respiratory and circulatory disorders*, which cause either a fluxion to and over-distension of the highly vascular gland, or congestive hyperæmia in it as a kind of safety-valve to the venous congestion of the intracranial organs. The occasion of these circulatory and respiratory disorders, according to the views of observers, may be either a chill (or the effect of cold air on the heated body, especially on the neck, or of a draught of cold water under the same circumstances), or it may be severe bodily strain such as climbing mountains, carrying heavy burdens (particularly on the head), and violent exertions, especially if they be put forth at great elevations or in a rarefied atmosphere.

¹ *Vide supra*, § 43.

Hahn points out¹ that goitre which was at one time of general occurrence among the female population of Luzarches, has disappeared almost entirely in recent times with the decline of the lace-making industry. His explanation of the fact is that the workwomen, who found employment at this industry from their early youth, were obliged to sit at their work with the neck thrown forwards, so that pressure was exerted on the cervical vessels and on the thyroid. Brunet² supports this opinion of the origin of goitre in general, on the ground of his observations in the department of Côte d'Or. On the other hand, Nivet, Halbron, Collin, Michaud, Utz, Chouet, and other French military surgeons, explain the epidemic outbreaks of goitre among the troops for the most part according to the second of the two ways mentioned: that is to say, as due to chills and strains of body, to which the soldiers are exposed in the course of their military exercises. Wilson's³ experiences in the Punjaub led him to a similar explanation of the cause of the goitre endemic there:

“Active occupation, necessarily so much more severe in hilly districts, seems to influence the production of this disease to a great extent, as is shown by its so frequent occurrence in those who lead a laborious life, or pursue active duties in a constrained position. . . . The effects of violent exercise upon the circulation and blood-vessels generally are well known, and it is only necessary, on this point, to refer to the relation of the thyroid gland to the large vessels of the heart, its remarkably large supply from them, and its dense capillary structure and consequent ready liability to enlargement from the dilatation of its vessels under the conditions produced by violent and prolonged exercise.”

This circumstance tells especially at the higher elevations, and that is the explanation, Wilson adds, why the male sex, being exposed to these bodily strains in a higher degree than the female, are more often affected with goitre in mountainous regions than the latter, who, in their turn, are more often affected in the plains. Chabrand is of opinion that even cretinism is referable to the same etiological factor—

¹ ‘Compt. rend.,’ 1869, lxix, No. 16.

² *Ib.*, 1869, lxix, Nr. 18.

³ ‘Med. Times and Gaz.,’ 1874, Dec., 693.

“à des perturbations profondes et fréquentes de la respiration et de la circulation,” these being due to the injurious influences above mentioned, to “passage brusque et fréquemment renouvelé d’une température froide à une température très-élevée, et vice-versâ, efforts, travail excessif, &c.”¹

It cannot be contested *a priori* that a hyperæmic swelling of the thyroid with dilatation of the vessels and other changes of the organ (hypertrophy and the like) subsequently ensuing, or, in other words, the formation of a goitre in the manner indicated, may take place and most probably does take place as a matter of fact ; or, at least, that a predisposing cause for the production of goitre is thereby furnished. But an explanation, after any rational fashion, of the *endemic* occurrence of that disease, and of cretinism as well, cannot be found therein ; and even for the epidemic outbreaks of goitre among soldiers particularly in French garrisons, and in boarding-houses, seminaries, and the like, the explanation does not seem to apply. All speculation as to the causes of endemic goitre and cretinism must be regarded as absolutely misdirected, which leaves out of sight the fact that both diseases have the marked character of local maladies and of local maladies confined within quite narrow circuits, and which seeks, in disregard of this fact, to find the disease-factors in such influences as are, so to speak, of a cosmopolitan kind. If this theory really possessed the general significance which observers have ascribed to it, then the diffusion of endemic goitre over the globe must needs be infinitely wider than as a matter of fact it is ; the epidemic occurrence of goitre—and this is the *punctum saliens* of the question—would not be limited to those regions where the malady is endemic for general reasons, but the same phenomenon would necessarily present itself in other regions as well, especially if they be mountainous, where there are the same entirely commonplace influences at work, such as catching chill, drinking cold water when the body is heated, over-exertion, and the like.

Several of the French practitioners have fully admitted the truth of this. Thus, Gouget, writing of the epidemic of goitre at Colmar in 1863, says that the troops previous to their arrival had gone through

¹ ‘Du goître et du crétinisme endémiques,’ Paris, 1864.

severe military exercises and had remained quite healthy all the while, the cases of goitre having occurred among them subsequently, or at a time when they were performing light garrison duty and were well lodged and fed. With reference to this theory, Viry and Richard state: "Nous sommes conduits à reconnaître, que la multiplicité des causes, auxquelles on a tour à tour rapporté l'apparition du goitre épidémique masque en réalité une grande incertitude touchant l'étiologie vraie de cette maladie."

There need be no question, as we have already remarked, that those factors which bring about enduring or frequently recurring hyperæmias of the thyroid, may furnish a predisposing cause of goitre-formation; and it is probably to this that we must refer the *predominance of the disease in goitrous districts among the female population*, in whom experience shows that the hyperæmic swelling of the thyroid stands in some connexion, which we cannot for the present define more closely, with physiological processes in the organs of generation (menstruation, pregnancy, childbed).

The larger number of authorities, who make any mention at all of the proportions of goitre in the male and female sex, confine themselves to stating that the disease occurs "preponderantly" or "almost exclusively" among women (Inglis, Addison, Bayers, and others for various parts of England, v. Franque for Nassau, Tourdes for Alsace, Mahue for the department of the Aisne, Challan for Kabylia, Bennet for Ceylon, Barton, Smith, Lane, and others for various parts of North America, and Duploux for Chili). Expressed in figures we find the proportion given by Morel for Sérécourt and by Manson for Nottingham to be 1 man to 11 women, and by Hallin for Faluh to be 1 to 12·3. The statistical data of the French Commission are not of much use, inasmuch as they relate to departments as a whole; according to them, the proportion for the whole of France is 1 male to 2·5 females, 1 to 2 for the departments most severely affected (Savoy, Hautes-Alpes, &c.), and 1 to 3 for those more slightly affected. Whether we may conclude from this that a higher prevalence in the male sex depends on the endemic intensity, I do not attempt to decide.

For the incidence of *cretinism* these differences in the two sexes are not discoverable, or at least the figures are too untrustworthy to warrant a definite conclusion. It seems as if the male sex were affected rather more.

§ 51. HOW FAR CAUSED BY THE DRINKING-WATER.

In the doctrine of the causes of disease, there is hardly any idea that has taken so deep a root, both in the popular belief and in the convictions of medical observers, as that *goitre and cretinism are caused by the use of drinking-water from particular sources*. This opinion is founded on the following experiences gathered from various parts of the globe: (1) that at places where the diseases in question are endemic, only those persons become the subjects of them who draw their supply of drinking-water from some particular spring, while those who get their water from other springs escape; (2) that goitre and cretinism have appeared as an endemic in villages after the opening of new water-supplies, the endemic reaching just as far as the water from such sources was used; and (3) that the endemics of goitre and cretinism have diminished in extent and finally died out after the suspected water-supply had been abandoned (or closed) and pains been taken to provide channels for another and innocuous drinking water.

The belief in the power of certain wells—"goitre wells" or "goitre springs"—to bring on goitre (and cretinism), goes as far back as our first information about goitre itself.¹ This idea took more definite shape when the belief arose, following in the wake of opinions expressed by Paracelsus and other practitioners and natural philosophers of the 16th century, that the property of goitre-wells to induce the disease was to be explained through their contamination by mineral matters; and since that time a very large number of observations have been brought together which have afforded grounds for believing that the origin of the disease is related in one or more of the above-mentioned ways to particular wells or springs.

Thus Boussingault writes from New Granada that a medical practitioner of Socorro, a town where goitre occurs in almost every house, obtained the supply of water for himself and his household from a rain-water cistern, and that every member of his numerous family kept free of the

¹ See the notes to the opening paragraphs of this chapter.

malady. Boussingault knew of another family in the highly goitrous town of Mariquita who had protected themselves from the disease by avoiding all drinking-water that had not been boiled. In St. Jean-de-Maurienne, according to Mottard, those of the inhabitants escape goitre and cretinism who avoid the suspected water from the well of Bourieux and obtain their supply from a large rain-water cistern that they have had erected. Moretin tells us that, some twenty years ago, a hamlet in the commune of Blegny (near Salins in the Jura) used to be much afflicted with goitre, but after the opening of a new spring the disease decreased very considerably; and the same thing had been observed in the village of Alleverd (Dept. Isère). Bergeret¹ remarks that at Saxon in the Vallais, goitre and cretinism had been very widely prevalent previous to 1835, but had almost entirely disappeared since that date coincidently with the giving up of the old water-supply and the opening of a new well. Conversely, Chatin mentions the development of an endemic of goitre in the villages of Fully and Saillon (Vallais) subsequent to the opening of a new water-supply; and the same thing is recorded by Aguilhon for the canton of Vertaizon (Puy-de-Dôme). Coindet and other Geneva practitioners have found that goitre had become strikingly less there since the town has been provided with water from the Rhone in pipes, and that the disease occurs only in those individuals who use the well-water which everyone used at one time, preferring it for its freshness. Germain² points out that the villages of Saint-Michel, Mornoz and Aigle-Pierre, near Salins in the Jura and situated on one side of the town, are much affected with goitre, whereas, in the communes of Pretin and Arsures on the other side of the town, only sporadic cases occur, the two groups of villages differing solely in respect that they get their drinking-water from different sources. The same observer mentions the extinction of endemic goitre in the small town of Nozeroy (Arrond. Poligny, Department Jura) on the opening of a new well. Reid³ informs us that the English residents

¹ 'Compt. rend.,' 1873, vol. 77, No. 13, p. 15.

² 'Bullet. de l'Acad. de méd.,' 1849, xv, 193.

³ Quoted by Saint-Lager, 'Etudes,' p. 194.

in Purneah protect themselves from the generally prevalent goitre by avoiding the water of the Coonee brook which flows past the town and by sending for water to the Ganges, three days' journey distant. In the report of the French Commission we find the following fact given:—In the town of Bozel (Tarentaise) there were counted in 1848 some 900 goitrous persons and 109 cretins in a population of 1472, while the village of St. Bon standing 800 metres higher was quite free from both diseases; but a water-pipe having been carried from that village to Bozel and this water having come into general use, the endemic decreased so remarkably that in 1864 there were only 39 goitres and 58 cretins, and no new cases were occurring.

Observations of the same kind are given by Fradenek¹ for Carinthia. There are some particularly striking statements as to goitres induced in Frenchmen and Italians, who purposely drank the water of "goitre wells," so as to escape military service. Observations to that effect, said to be well authenticated, are given by Saint-Lager² for Savoy; and Lombroso,³ speaking of Lombardy, says: "A Cavecurta vi ha la, 'fonte del gozzo,' ove sogliono andare i giovani all' epoca della coscrizione onde acquistare in quindici giorni quel difetto che li sottrae dal servizio."

Noteworthy as these and many other observations are, there are not wanting others which serve to place in a somewhat questionable light the conclusions as to the pathogenesis that have been drawn therefrom. As we have already seen,⁴ goitre and cretinism have appeared as a new thing in some localities, just as they have disappeared from others on improvement of the hygienic conditions, without any obvious change whatsoever occurring in the drinking-water. Again, it has been pointed out by many observers that, of a number of villages all in one neighbourhood, some are afflicted with goitre and cretinism, while others, drawing their water-supply from the same source as the former, enjoy an absolute immunity from these diseases; this has been observed by Rösch in Würtemberg, Rüdel and others in Middle Franconia, Schaussberger in Upper and Lower Austria, Meyr in the circle of Kronstadt (Transylvania),

¹ L. c., p. 456 ff.

² P. 191.

³ P. 16.

⁴ *Supra*, pp. 153, 180.

Maffei in the Salzburg Alps, Miral-Jeudy, in Clermont-Ferrand (Puy-de-Dôme), Evans in Tirhoot, Bramley in Nepaul, and Humboldt in New Granada. It has been shown also by Gouget, Morelle, Fleury, Viry and Richard, Muller and Michaud, that the epidemic outbreaks of goitre in French garrisons cannot be brought into any casual connexion with the water, inasmuch as those of the troops who were the victims of the epidemic obtained their drinking-water from the same spring that supplied the unaffected barracks as well as the civil population, none of the latter having had any part whatsoever in any of these epidemics.

There are, indeed, explanations offered by way of resolving these contradictions in what I may call the drinking-water theory, or by way of weakening the force of the objections that have been brought against it. Saint-Lager in particular has with much skill endeavoured to prove that, in those cases where decrease or extinction of endemic goitre and cretinism has been achieved through improved hygienic conditions, there has probably been some change at the same time in the particular substances contained in the drinking-water; and that the unequal distribution of the malady in the various places deriving their water-supply from one source, is to be explained by the fact that the same stream may contain different kinds of mineral substances at various stages of its course, owing to washings of the soil or other additions. However, this does not by any means serve to reconcile the contradictions; and although one should hesitate, in view of the many positive facts, to give up the drinking-water theory of the production of goitre and cretinism as absolutely untenable, yet a certain scepticism in this question is called for, all the more so that no one has hitherto succeeded in detecting in the suspected "goitre wells" or "goitre springs" any one uniform thing either of mineral or organic nature, common to them all and occurring in them only.

It was formerly held by some observers, and it is still a popular belief in certain mountainous parts of South America (New Granada, Chili¹ and Peru), that *goitre is a consequence of drinking snow-water or glacier-water*, being caused,

¹ Duploux refers to it as the belief still universally current in Santiago.

as Boussingault¹ and others² have assumed, by the small amount of atmospheric air in the water.³ This doctrine, already overthrown by Foderé,⁴ requires no further refutation when we consider that the disease occurs at innumerable points of the globe, where there cannot be the remotest thought of drinking-water of that kind, and that the use of distilled water, even for a considerable period, has been shown to be quite unattended with harm.

The most popular of all these doctrines has been that which makes the peculiar property of causing goitre and cretinism to depend upon *the abundance of lime salts (carbonate and sulphate of lime, but particularly of magnesia) in the drinking-water*. The fact that water rich in lime and magnesia is exceptionally often drunk in regions where goitre and cretinism are endemic, will not be contested, inasmuch as we have seen that those diseases occur, if not exclusively, yet for the most part upon limestone and dolomite soil ; but it is by no means proved that the partaking of water of that kind is actually the cause of these diseases. On the other hand, there are facts, both positive and negative, which tell against any dependence of the pathogenesis on that factor.

Referring to the amount of lime in the drinking-water of localities in Würtemberg, both those affected by goitre and cretinism and those exempt from them, Rösch⁵ has the following : “ Many of the streams, particularly in districts and localities where goitre and cretinism are endemic, contain as much gypsum as cold water can dissolve or retain in solution. Notwithstanding this, goitre and the cretinous degeneration do not exist at every place where the water contains gypsum ; for example, they do not occur, or they occur only to an insignificant extent, at Gaildorf, Murrhardt, Botenheim, and other villages. At Tübingen the constituents of the water

¹ He became convinced afterwards that the theory was untenable.

² Rendu, writing of Brazil, says that this opinion is still held by Dr. Faivre.

³ Somewhat allied to this opinion is the doctrine given out by Rozan (‘ Mem. de méd. milit.’ 1863, x, 357), according to his experiences in Briançon, and adopted by Lombard (‘ Étude sur le goitre et le crétinisme endémiques,’ Genève, 1874), to the effect that respiration in rarefied air, or in other words, *a lesser amount of oxygen in the air*, is a cause of goitre. The rapidity of the respirations, as we know, is in proportion to the amount of oxygen in the air.

⁴ L. c., p. 26.

⁵ L. c., p. 213.

are almost the same in all the wells, and yet goitre and cretinism occur only in the lower part of the town belonging to the Ammerthal. On the other hand, goitre (along with cretinism) is found at a number of places where the water contains either no gypsum or the merest trace of it, as in the Glatthal, the Nagoldthal, and on the Lake of Constance, precisely as it is found where there is gypsum in the water." The same conclusion has been come to by Maffei¹ as regards the condition of the drinking-water in the Salzburg Alps. Klebs also, who formerly² laid particular stress on gypsum in the drinking-water as a cause of goitre and cretinism, was obliged afterwards³ to admit that the water in the Salzburg centres of goitre and cretinism was almost free from mineral constituents. Rossknecht observes that the water supply of the very goitrous commune of Hammereisenbach in Baden comes out of granite and is absolutely free from saline ingredients; and Weber⁴ points out that in Mannheim, where the water is rich in lime, the goitrous are 0.77 per cent. whereas in Heidelberg, where the amount of lime is very small, they are 5.72 per cent. In the valley of the Neckar in Hesse, where the two diseases are endemic, we learn from Müller that the water used for culinary purposes is chemically almost pure, whereas in the side valleys which use the same water, neither goitre nor cretinism is met with. According to Herberger, the drinking-water of Rheinzabern in the Palatinate, where goitre and cretinism are endemic, contains mere traces of lime and magnesia. In the village of Ridgmont in Bedfordshire, goitre is endemic although the water is free from lime, whereas in localities adjoining where the water is rich in lime, the malady does not occur.⁵ Goitre is endemic also at Bolton (Lancashire) notwithstanding the absence of lime in the water.⁶ In Switzerland, as Amsler shows, goitre is much commoner in localities where the water is poor in lime than in those where the amount of lime is great. The same fact had previously been demonstrated by Zschokke in connexion with the distribution of the disease among the villages in the valley of the Aar, and we have

¹ L. c., p. 160.

² 'Arch. für exper. Pathol.,' 1874, ii, 87.

⁴ L. c., p. 31.

⁵ Blower, l. c.

³ 'Studien, &c.'

⁶ Black, l. c.

corresponding observations on the goitre and cretinism of the Canton Vallais. In the Champagne, where the water drunk is much impregnated with lime, endemic goitre is unknown¹. The three wells in Chambéry, which are considered to be most under suspicion, contain only traces of sulphate of lime and magnesia.² At St. Jean, St. Sulpice, St. Rémy, and other places in the Lower Maurienne, which are the head-quarters of the endemic of goitre and cretinism there, the drinking-water is much purer, that is to say, freer from mineral matters, than in the Upper Maurienne where neither goitrous persons nor cretins exist.³ How slight an influence the abundance of lime in the water has upon the occurrence of goitre in Italy is shown by Sormani,⁴ in the fact that Bologna, Florence, Leghorn, and Rome, where the water in general use is hard, have goitre only as a rarity, and in the other fact vouched for by Professor Taramelli, that in Vicenza and the Abruzzi, being the provinces of Italy where lime most abounds, the malady occurs only to the very smallest extent. In many parts of North America, which are or have been affected with goitre, such as Bennington, (Verm.) Pittsburg, and Fort Dayton, the drinking-water, as Barton informs us, does not contain lime, whereas lime-salts predominate in the water of many parts of Pennsylvania where the disease has never been endemic.

As regards *magnesia*, none of it has been found by Nièpce in the drinking-water of Bourg d'Allevard, Sassenage and other places near Grenoble, or in the water used in many of the goitrous villages of the Hautes-Alpes and Basses-Alpes. In the commune of Coise there are two wells, one of which is looked upon as causing goitre and the other as curing it; analysis of the water in each of them gives the following results:

	No. 1.		No. 2.
Carbonate of lime .	0·166	.	0·680
Sulphate of lime .	0·049	.	0·027
Chloride of calcium .	0·009	.	0·028
Chloride of magnesium	0·000	.	0·035

¹ Robinet, 'Gaz. des hôpit.,' 1863, Jan., p. 15.

² Bonjean, 'Gaz. méd. de Paris,' 1851, p. 135.

³ Fodéré, l. c., p. 27.

⁴ L. c., p. 164.

Chevalier has shown that the water in Aosta, Villard, (Tarentaise) and Valnaveys is free from magnesia, whereas that substance is contained in the water of the Graisivaudan valley, which is free from cretinism. Maumené says¹ that at Rheims, where goitre used to be endemic, there is not a trace of magnesia to be found in the soil or in the well-water. Dejean has examined the drinking-water from localities in the Jura where goitre is endemic, and has found that the amount of magnesia is least in the Canton Voiteur, which is actually the most goitrous region in the department. Agreeing herewith is the observation of Moretin that magnesia is found everywhere in the water of the Seille, equally at goitrous localities and at those where the malady does not occur, while there is no definite relationship discoverable between the amount of magnesia in the water and the amount of the disease at each place. In like manner Tourdes² found magnesia in the drinking-water of a few goitrous and cretinous villages of Lower Alsace, but the same constituent showed itself also in the drinking-water of other places in the department, where the maladies were either on the decline or were quite unknown. In the town of Rodez (Dept. Aveyron), where neither goitre nor cretinism occurs, there is five times as much magnesia in the water as at the goitrous and cretinous spots in the valley of the Isère;³ also in Noyon, where the water used is very rich in magnesia, there is no endemic goitre.⁴ Demortain has examined the water at several goitrous spots in the plain of Lombardy, and found it to be absolutely free from magnesia.⁵ In Faluh, the single locality of Sweden where goitre is endemic, a kind of drinking-water is used which has not its like in the whole country for chemical purity.⁶ In the districts of the Punjaub which are subject to goitre and cretinism, the drinking-water has so little of mineral ingredients that it may be described as absolutely pure.⁷ On the other hand both diseases are unknown on the western littoral of Mexico (Guaymas and Mazatlan), although the water used there contains magnesia.⁸

¹ 'L'Institut,' 1850, No. 870, p. 282.

³ Blondeau, as quoted by Moretin, p. 42.

⁵ 'Gaz. hebdomadaire de médecine,' 1859, p. 709.

⁷ Wilson, l. c.

² L. c., p. 18.

⁴ Guilbert, l. c.

⁶ Berg., l. c., p. 47.

⁸ Lucas, l. c.

Allied to the theories here discussed (and shown to be untenable) which assert the origin of goitre and cretinism from the use of drinking-water peculiarly rich in lime or magnesia, there are certain other views, belonging to the same order, which have been put forward recently. Such is the conjecture of Schwalbe¹ that *a deficiency of chlorides in the water, especially of common salt, causes goitre and cretinism*. This conjecture is supported by the observation of Eulenberg² that in the villages of the Coblenz circle where goitre is endemic the water is strikingly poor in chlorides, while in the villages exempt from the malady the chlorides in the water are abundant; it is supported also by Demortain's statement that a notable absence of chlorides is found to exist in the drinking-water of the goitrous districts of Lombardy, and by the analysis of waters in some other centres of the malady. Apart from the objection justly taken by Virchow,³ that it is scarcely credible "that an active, nay even an irritative process can be induced by the mere absence of a substance rather than by some positive substance or combination," it is further impossible to understand how any secret power to call forth such effects can reside in the common salt of drinking-water, present for the most part in merely minimal quantities, when every day we introduce into the organism enormous quantities of salt with all our food—such a power as must be able, if we are to trust the accounts of the wilful production of goitre by drinking the water of "goitre-wells," to produce its effects within the space of a fortnight.

Saint-Lager's conjecture, based upon his opinion that goitre and cretinism occur only in those regions where there are metals in the soil, is to the effect that *the presence of metallic substances in the drinking-water, particularly sulphate of iron, is the cause of the disease*. In somewhat the same sense McClelland had already propounded the question whether perhaps the presence of copper in the soil might not impart goitre-producing properties to the water. Low also has recently called attention to the fact that the drinking water in all the goitrous localities of Yorkshire is distinguished by its large amount of iron and alum.

¹ L. c., p. 31.

² 'Archiv für gemeinschaftl. Arbeiten,' 1860, iv, 347.

³ 'Geschwülste,' iii, p. 59.

Saint-Lager speaks with the reserve characteristic of an impartial inquirer: "Let us take care," he says at the end of his discussion,¹ "not to go too far; the sulphate of iron is for the present only an accused party, until such time as culpability can be proved of it in a conclusive manner." His experiments with sulphate of iron given to dogs have miscarried through accidental causes. Still more dubious is McClelland's expression of opinion:

"The noxious principle in the waters of Alpine limestone," he says,² "is a subtle combination [of various minerals] derived perhaps from the strata of the rock which are called by miners 'copper slate.' They are so distinguished from the quantity of metals which they contain, particularly the ores of copper. In describing the locality of the springs, which supply those villages where the inhabitants suffer most from goitre, they may be said to be generally derived from the strata in question, or, at least, from the lower beds of limestone near the junction where it rests on clay slate. But whether there be any other strata," he adds,³ "capable of yielding this peculiar contagion than those we have described, and whether the water is the only medium by which it is conveyed, are points which still remain to be determined."

The objections to the theory, as we have already seen, (p. 178), are, on the one hand, that goitre is endemic in localities where not a trace of iron pyrites can be detected in the soil, while the malady does not occur in other regions whose soil is unusually rich in that mineral; and, on the other hand, that the long-continued therapeutic use of salts of iron, including sulphate of iron in mineral waters, has never, so far as we know, been followed by the development of goitre. It ought not to be difficult to verify the hypothesis put forward by Saint-Lager by means of experiments on such animals as are otherwise prone to goitre, and possibly also by observations in medical practice.

I have to mention in conclusion the view of Maumené⁴ according to which *fluorine gives rise to goitre*. His conclusion was derived from the general diffusion of that element in the soil of goitrous regions (especially in the Pyrenees where he had travelled). To test it he experimented on a cat, which he plied with fluoride of potassium for five months; towards the end of that period a swelling developed in the neck, but the cat then made its escape, and when it was re-

¹ 'Études,' p. 454.

² 'Sketches of the Med. Topogr., &c., of Bengal,' Lond., 1859, p. 92.

³ *Ib.*, 110.

⁴ 'Compt. rend.,' 1866, févr. 19 ('Arch. gén. de méd.,' 1866, avril, 497).

captured three years after, the tumour, which was still there, was found on dissection by Professor Gaillet of Rheims to have nothing to do with goitre. Saint-Lager's experiments on animals with fluoric acid have resulted negatively.¹

§ 52. THEORY OF DEFICIENCY OF IODINE IN THE AIR AND WATER.

A short-lived opinion was that advocated by Chatin² (before him by Prevost and Maffoni,³ and after him by Marchand⁴ and Fourcoul)⁵ to the effect that the cause of goitre and cretinism lay in the *absence of iodine in the drinking-water and in the air*. Chatin pointed to his numerous inquiries which showed that wherever the amount of iodine was relatively large, as in the basins of the Seine, Yonne and other rivers, the two diseases were unknown; that they both became prominent with a smaller amount of iodine, as in the Rhone valley; and that this inverse ratio obtained with even greater force in the valley of the Isère, and most of all in the very intense goitrous and cretinous localities of Tarentaise and Maurienne. Against this theory there is first of all the fact, admitted by Chatin himself, that in mountainous regions the quantity of iodine is inversely as the height of the place, while the greatest prevalence of goitre and cretinism occurs at the more deeply situated spots, decreasing in proportion as we ascend. The inquiries made by Dejean, Germain, and Moretin into the amount of iodine in the water of various places in the Jura have resulted in showing that it is nearly zero everywhere, whether in the villages with endemic goitre or in those without it. Niepce has been able to make out the presence of iodine sometimes in considerable quantity, in the air, the water, and the vegetation of the plain of the Po, in several localities of the department of Saône-Loire, in a few villages of the Val d'Aosta, and in the valley

¹ 'Études,' p. 457.

² 'Compt. rend.,' 1850-52; 'Gaz. des hôpit.,' 1852, Nr. 4 ff; 'Compt. rend.,' 1853, i, 652.

³ 'Atti dell' acad. med.-chir. di Torino,' 1846, ii, 453.

⁴ 'Compt. rend.,' 1850, ii, 495.

⁵ *Ib.*, 1851, ii, 518.

of the Isère, all of which are subject to endemic goitre and in part also to cretinism. Bebert¹ found a not inconsiderable amount of iodine in several springs in the valley of the Maurienne, corresponding to localities where goitre and cretinism were very widely prevalent. Casaseca² affirms that in Cuba there is not a trace of iodine discoverable either in the air or in the water, and yet that island, like most of the Antilles, is quite free from goitre. Saint-Lager³ mentions a very notorious "goitre-well" at Beaulieu in the department of the Oise, which contains a large amount of iodine as well as of iron; and he adds the remark that the springs most impregnated with iodine are those that come from a soil particularly rich in vegetable detritus and peat, and that these are just the springs most notorious for their goitre-producing properties. The only other point that I shall advert to is that the theory of Chatin would not so much serve to explain the cause of goitre and cretinism, but rather serve, if it be well-grounded, to show that the actual disease-factor itself widely spread over the globe, is only prevented from becoming potent because it is neutralised within the human body by the iodine introduced (in the most minimal quantities) into the organism along with air and food. It would then remain a question, and a very doubtful one, whether iodine has a prophylactic power against goitre, as well as a curative.

§ 53. A PAIR OF INFECTIVE DISEASES DUE TO A MORBID POISON.

The absence of results to all these inquiries about the genesis of goitre and cretinism—inquiries which have extended to every influence perceptible to the senses that could be brought into the consideration of the question before us—warrants, I will not say compels the conclusion that in those diseases we have to do with a specific agent, a *veritable morbid poison*, and that endemic goitre and cretinism *have to be reckoned among the infective diseases*. This doctrine finds a certain support in two things: on the one hand the fluctuations

¹ Quoted by Vingtrinier, p. 28.

² 'Compt. rend.' 1853, ii, 348.

³ L. c., p. 240.

in the amount of the sickness, which we have had frequently occasion to notice, and for which there is no sufficient explanation to be found in the states of the atmosphere or of the soil or of hygiene; on the other hand, the epidemic outbreaks of goitre, for which the theory of infection, as Saillard, Viry and Richard, Thibaud and other French military surgeons have recognised and said, is hitherto the only one that affords an explanation, and an explanation that accords most nearly with the facts of the case.

Humboldt was the first, so far as I know, to express this opinion as to the nature and the cause of goitre and cretinism, the hypothesis having subsequently received the adhesion of Vest,¹ Bramley,² Troxler,³ Gugger,⁴ Schausberger,⁵ Virchow,⁶ Moretin,⁷ Vingtrinier,⁸ Morel,⁹ Berkowski,¹⁰ Köberle,¹¹ the French Commission, Nivet,¹² and myself (in the first edition of this work). As to the nature of this goitrous and cretinous virus, and its means of conveyance, it is impossible to form a well-grounded opinion. Its existence and development would appear to depend upon certain definite kinds of soil, such as a soil containing dolomite rock, and it would appear to occur principally in water, perhaps associated also in some circumstances with plants or suspended in the air. Whether its nature is organic or inorganic is a question that evades our answering. At all events it has nothing in common with malaria, as Tourdes, Fayrer, Ance-lon,¹³ and others would seem to say, inasmuch as goitre and cretinism are endemic, and endemic even to a very considerable extent, in places that enjoy the driest of situations and

¹ 'Salzburg. med.-chir. Ztg.,' l. c., and in Fradenek, l. c.

² 'Calcutta Transact.,' l. c.

³ 'Der Cretinismus, &c.,' 1836.

⁴ 'Oest. med. Jahrb.,' 1839, Nste. Folge, xix, 85.

⁵ 'Oest. med. Wochenschr.,' 1842, 1091.

⁶ 'Verhandl. der Würzb. Gesellsch.,' 1852, ii, 268, and 'Gesammelte Abhandl.,' 958, 968.

⁷ L. c., 57.

⁸ L. c., 18, 39.

⁹ 'Annal. med.-psychol.,' 1854, Oct., and 'Arch. gén. de méd.,' 1864, 173.

¹⁰ L. c.

¹¹ 'Essai sur le crétinisme,' Strasb., 1863.

¹² 'Gaz. hebdom. de méd.,' 1874, 55.

¹³ Ib., 1857, 62.

are most free from all marshy exhalations; while on the other hand, there are large malarious tracts of low country, especially along the course of great rivers, and not a few of them in closest proximity to goitrous and cretinous spots, which are perfectly free from both of these maladies. Klebs, who had at one time¹ characterised the doctrine of the infective nature of endemic goitre and cretinism as a "theory of mystery" is now of opinion² that he has found the morbid poison in the form of minute animal or vegetable structures, called by him "naviculæ" from their shape; these he has detected in the well-water of several goitrous and cretinous districts of Salzburg. Whether the "naviculæ" do not involve an even darker mystery must remain at least a question. Rüdel has been unable to find these "naviculæ" in suspected wells which he examined.

§ 54. RELATION OF CRETINISM TO GOITRE OF THE PARENTS.

To conclude these inquiries I have still to justify an assumption with which I set out, the assumption, namely, *that goitre and cretinism, being a pair of disease-types closely related in their pathology and etiology, are to be regarded as different expressions of a single morbid process.* This idea is fully borne out not only in the territorial distribution of the two diseases alongside each other, nor only in the fact that by far the most of cretins (three-fourths of them at least) are at the same time goitrous, but above all in the fact which I now come to, that cretinism arises hereditarily or congenitally where the parents are goitrous. No doubt we find large regions of goitre in which cretinism does not occur or occurs only sporadically; but, as Virchow very justly remarks, "at every place where there is cretinism associated with conditions of the locality, goitre also is endemic; . . . and we cannot but trace both diseases to the same kind of influence, goitre being regarded as the result of a feebler working of the noxious power, and cretinism the issue of a stronger working." Almost all observers, both old and new,

¹ 'Arch. für exper. Pathol.,' l. c.

² 'Studien über die Verbreitung des Cretinismus,' 1877.

including Fodéré, Iphofen, Rösch, Marchant, Tourdes, Morel, Niepce, Bouchardat, and the French Commission have adopted this view.¹ The opinion of Ackermann, Maffei, and the Sardinian Commission, of Ferrus, Moretin, and Köberle, that the endemic prevalence of the two diseases beside each other is no more than an accidental coincidence, is one that we are perfectly warranted in rejecting as baseless.²

The most decisive evidence in this question is the fact that *by far the larger number of cretins are the offspring of goitrous parents*,³ whether the latter had been at the same time affected with cretinism or not.⁴

The following figures are derived from inquiries into the inheritance of cretinism made by the Sardinian Commission.⁵ Of 4899 cretins, there were 1631 in which neither parent suffered from goitre or cretinism, the parents of the remaining 3268 having been affected as follows :

	Goitre.	Cretinism.	Goitre and cretinism.	Uncertain.
Fathers .	962	51	106	396
Mothers .	1281	43	66	363
	2243	94	172	759

According to these figures, the proportion of cretins whose parents (not reckoning the 759 uncertain cases) were notoriously the subjects of goitre, or of cretinism, or of both, was 51 per cent., and those whose parents suffered from goitre only were about 50 per cent.; but

¹ "To discover the causes of goitre," says Marchant ('Observ. des Pyrenées,' Paris, 1842), "is also to discover the causes of cretinism." Morel says: "Goitre is the first stage on the road leading to cretinism;" and the opinion of Niepce is to the like effect: "Goitre is the first degree of a degeneration of which cretinism is the final result."

² The weight attaching to these dissentient opinions may be estimated from the following statement by the Sardinian Commission (p. 43): "Si l'on considère, qu'il se trouve des crétiens entièrement privés du goître, que le degré du crétinisme n'est pas toujours en raison directe de son volume, qu'enfin on rencontre des individus portant un goître volumineux sans présenter le moindre indice de crétinisme, il est permis de conclure que le goître ne constitue pas un symptôme essentiel, mais qu'il forme une concomitance purement accidentelle de cette triste dégénération."

³ "This heredity," says Tourdes, "is one of the clearest proofs of the community of type which characterises the two affections."

⁴ The marriage of persons suffering from cretinism is on the whole very rare, and those unions in which there is cretinism on one side are mostly unfruitful.

⁵ L. c., p. 162-3.

there is no doubt that the proportion is even greater, as the statistics of the following authorities tend to show :

Table showing the Goitrous Parentage of Cretins.

Total number of cretins.	Goitrous parentage.	Authority.
58	42	Marchant.
20	14	Anzouy.
75	52	Roque.
12	9	Billiet.
161	147	Fabre.
35	30	Ménestrel.
361	294 = 81.4 per cent.	

Fodéré was the first to recognise this causal connexion between goitre of the parent and a cretinous malady in the offspring ; and the fact has received a large amount of confirmation, besides the statistical support already mentioned, in the experiences of Roulin, Tourdes, Grange,¹ Morel,² Garrigou, McClelland,³ Köstl,⁴ the French Commission and others, the conclusion being expressed in the words of Fabre,⁵ “le goître est le père du crétinisme.” The fact that in many regions goitre and cretinism are prevalent together, while in others goitre only is endemic, indicates that the “parental” power of goitre is dependent on still other influences exerted on the foetus by the progenitors, whereby the development of the former is modified or a disposition to cretinism induced. It is extremely doubtful whether cretinism ever arises spontaneously, that is to say, without a congenital predisposition, and merely from the intensified action of those influences which suffice in a lesser degree to produce goitre. In the whole of the extensive literature of cretinism there is not a single case to be found in which a child born of parents free from goitre or cretinism, and in a locality exempt from them, has become cretinous on being brought in infancy or youth to live in a cretinous region. Niepce and several others absolutely deny the possibility of such an occurrence, adhering to the dictum of Morel : “on devient goîtreux et l’on naît crétin.” That

¹ ‘Compt. rend.,’ 1849, ii, 696.

² ‘Arch. gén. de méd.,’ 1864, 173, 1865, juill., 5.

³ ‘Topography, &c.’ 118.

⁴ L. c., 107.

⁵ L. c., 257.

there may be a conveyance of the endemic morbid poison to the offspring from healthy parents is shown by the observation that in cretinous regions children become cretinous by no means unfrequently when the parents are subject to neither goitre nor cretinism. The most interesting of these cases are those in which healthy parents have migrated from localities free from goitre and cretinism into cretinous centres, cretinism appearing among the offspring born to them there although they themselves had not become goitrous. A case of that kind is recorded by Virchow¹ on the information of Dr. Schierlinger of Reichenhall: "an official transferred thither whose previous children had been healthy, had 'Fexe' born to him during his residence in Reichenhall, while the children born after he was transferred back were healthy as before." Another case is given by Morel, that of a man travelling a stud-horse, who came with his wife to Rozières-aux-Salines, one of the chief seats of cretinism in the department of the Meurthe, and there had a cretinous child born to him, although his former children had been healthy. On the other hand the observation has never been made that goitrous parents migrating from an endemic region of cretinism into a healthy region, have there had cretinous children. In the majority of cases the first traces of the inherited disposition are observable in the child at the second or third year, and perhaps never so late as the fourth year. But, that cretinism may exist even at birth, follows from a case given by Virchow,² and from the following statement of the Sardinian Commission:³ "Le crétinisme ne se comporte pas toujours comme les maladies héréditaires; quelquefois en effet ce n'est plus seulement la disposition au crétinisme, qui se transmet des parents aux enfants, mais bien le vrai crétinisme."

Cretinism having once appeared in a family may propagate itself through several generations, of which fact there are numerous instances. There is very nearly unanimity among observers that *goitre is transmitted by heredity*; and it can hardly be doubted, also, according to the statements of Rösch and others, that goitre is sometimes, although rarely congenital.

¹ L. c., ii, 267.

² L. c., p. 260.

³ L. c., p. 194.

CHAPTER V.

ERGOTISM.

§ 55. HISTORICAL NOTICES OF ERGOTISM.

Under the name of “saint’s fire” (*ignis sacer*, *ignis S. Antonii*) the mediæval chroniclers have described a kind of epidemic disease, observed mostly in France, which was characterised by intense pain and gangrenous destruction of the skin, the gangrene extending not unfrequently to the other soft parts and to the bones, and thereby leading to loss of the extremities. In many of the epidemics the mortality was frightful. There is no longer any doubt, after the inquiries by Read,¹ by the Commission appointed by the Medical Society of Paris² (which consisted of Jussieu, Paulet, Saillant, and Tessier), by Sprengel,³ and by Fuchs,⁴ that in all these epidemics of “saint’s fire” we have to do with *Ergotismus gangrænosus* (*Brandseuche*). The chroniclers of the period had applied the term in a somewhat special sense, whereas the practitioners of the Roman Empire, the Arabians and the mediæval practitioners had included under the general notion of “*ignis sacer*” a variety of diseases, (anthrax, malignant erysipelas, &c.), distinguished by intense burning and ulcerous or gangrenous destruction of skin. We may conjecture that ergotism was included under that head, inasmuch as the statements of Pliny, Galen, and other authors of antiquity lead us to infer that malignant diseases due to the partaking of damaged or unwholesome grain had been by no means rare.

¹ ‘Traité du seigle ergoté,’ Strasb., 1771.

² ‘Mém. de la soc. roy. de méd. de Paris,’ Année 1776, Paris, 1779, p. 260.

³ ‘Opuscula academica,’ Lips., 1844, p. 89.

⁴ In ‘Hecker’s wissenschaftl. Annal. der Heilkunde,’ 1834, xxviii, 1.

The following passage of Galen,¹ in which he draws attention to the poisonous properties of moulds growing on corn, is especially deserving of attention:

“Edu lia vero prava intelligo, tum quae ex natura talia sunt . . . atque etiam quae bona sunt ex natura, sed ob putredinem quandam praedictis aequale aut majus saepenumero vitium acceperunt, ut hordeum ac triticum et aliae omnes fruges frumentariae, partim ob temporis longitudinem ad putridam dispositionem deductae, partim situ quodam repletae, quia prave repositae sunt, *partim ex prima generatione a rubigine (ὑπ’ ἐρυσίβης) vitiatae*. Tales igitur cibos etiam nunc plerique comedere coacti prae fame, alii *febris putridis ac pestilentibus* mortui sunt, alii pustulis scabiosis et leprosis correpti.”

We have no means of judging whether the *convulsive form of ergotism* (*Kriebelkrankheit, raphania*) had occurred in ancient and mediæval times, along with or independently of the gangrenous form. Of that disease our first accounts date from the sixteenth century, and it is noteworthy that they come from countries which have been less frequently the subject of gangrenous ergotism, and not from France where the latter form of disease has been most prevalent from the earliest times down to the most recent. In a few epidemics, however, both forms of the malady have been observed together. As a basis for my sketch of the geographical and historical relations of the disease I prefix the following chronological table of all the more considerable epidemics of ergotism known to me, omitting those in which merely isolated cases are spoken to:

*Chronological Table of Epidemics of Ergotism.*²

Year.	Country.	Authority.
591	*France (Limoges) . .	Gregor Tur., Hist. Franc., x, 30.
857	*Germany (Rhinel.) . .	Annal. Xant. in Pertz, Monum., ii, 230.
945	*France (Paris)	Frodoardi, Chron., ib., iii, 389.
993-4	*France (Perigord, Angoumois, Limousin)	Radulphi, Hist., ib., vii, 61, Bouquet, Collect., x, 147, 318, 361, Mezeray, Hist., ii, 5.

¹ ‘De differentiis februm,’ lib. i, cap. iv, ed. Kühn, vii, 285.

² Where a star (*) is prefixed, the epidemic is ergotismus gangrænosus; the absence of a star indicates raphania or convulsive ergotism. A cipher (0) indicates that both forms occurred together.

Year.	Country.	Authority.
996	*France (Lorraine) ...	Calmet, Hist. de Lorraine, ii, Praef. xi.
999	*Spain (Leon)	Villalba, Epidemiol., i, 40.
1039	*France	Radulphi, Hist., l. c.
1041-2	*France	Hugo, Chron. in Pertz, Monum., viii, 403; Bouquet, xi, 145.
1085	*France (Lorraine) ...	Königshofen, Chron., 302; Bouquet, xii, 465.
1089	*France (Lorraine, Dauphiné)	Sigberti, Chron. in Pertz, vi, 366; Bouquet, xiii, 159, xiv, 141.
1089	} *Netherlands (Flan-	} Chron. van Vlaendern, i, 114, 567.
1092	ders)	
1094-5	*France	Bouquet, xii, 427, xiii, 260.
"	*Netherlands (Flan-	Chron., i, 118, 119.
"	ders)	
1099	*France (Dauphiné)...	Chron. Ursperg., 177.
1109	*France (Orleans, Chartres, Dauphiné)	Bouquet, xii, 690, 708, xv, 148.
1110	*England	Short, On the Air, Weather, &c., 108 (untrustworthy).
1125	*France	Chron. Ursperg., 206.
"	*Germany (Silesia) ...	Henschel, Med. in Schlesien, Bresl., 1837, 38 (untrustworthy).
1128-9	*France (very widely spread)	Bouquet, xii, 283, 780, xiii, 269, 328, 495, 582, xiv, 18, 234.
"	*Netherlands (especially Utrecht)	Chron. van Vland., i, 175.
"	*Germany	Accord. to Ozanam (very untrustworthy).
"	*England	Short, l. c., 115.
1140-1	*France (Paris and other places)	Bouquet, xii, 558, xiii, 27, 501, 582, xiv, 20.
1151	*France (very widely spread)	Ib., xiii, 275.
1180	*France (Lorraine) ...	
"	*Spain	Villalba, i, 47.
1205	*France	Annal. Elnonens. in Pertz, Monum., v, 16.
1214-5	*France	} Villalba, i, 53-57.
"	*Spain	
1230	*Majorca	Ib., 58.
1236	*France (Poitou)	Fuchs, 77, § 49.
1254	*France (Marseilles)	Foderé, Leç. sur les épidémies, ii, 45.
1256	*Spain. (Salamanca, &c.)	Villalba, i, 62.
1347	*France (Bretagne)...	Chron. Briocense, in Fuchs, 78, § 53.
1373	*France	Mém. de la soc. de méd., 270, 274.
1460	*Sicily (Trapani, Palermo)	Ib., 274, and Renzi, Sul clavismo cancr., Napoli, 1841.
1486	*Germany (very general)	Fabricius, Annal. urbis Misnionae and var. other chron. ¹
1565	*Spain	Villalba, i, 178 (untrustworthy).

¹ The disease is here referred to under the name of Scharboek (scurvy).

Year.	Country.	Authority.
1581	Germany (Lüneburg)	Rousseus, Miscell. in Schenck, Obs. med. lib., vi, Fft. 1665, 830.
1587	Germany (Sudetic Mountains)	Schwenckfeld, Theriotroph. Silesiae. Lignic., 1605, 334.
1590	*Spain	Villalba, i, 208 (after Fragoso).
1592	Germany (Silesia) ...	Schwenckfeld, l. c.
1595-6	Germany (Westphalia, Rhine, Brunswick, Hanover, Holstein, Saxony, Hesse, Breisgau, &c.)	Bericht von der Krampfsucht., Marb., 1597, a Bra in Foresti, Observ. xx, Lugd. Bat., 1595, 414; Wier, Obs. med., ii, § 1; Opp. Amstelod., 1660, 946.
„	Holland (Gelders, Friesland, &c.)	A Bra, l. c.
1600	Germany (Upper Hesse)	Horst, Opp. Norimb., 1660, ii, 422.
1630	*France (Sologne) ..	Thullier, Journ. des Sçav., 1676, iv, 79.
1648-9	Germany (Saxon Voigtland)	Leisner, Trakt. von der Krampfsucht. Plauen, 1676; Buddaeus, Cons. med. von der Krampfsucht. Budiss., 1717; Hoffmann, Med. rat. syst., t. ii, p. ii, cap. ix, § 15.
1650	*France (Guyenne, Sologne, Gatinois, Perault)	Thuillier, l. c.
„	*Switzerland (Bern, Zurich, Lucerne)	Lange, Descr. morbi ex esu clavor. secal., &c., Luz., 1717.
1660	} *France (Sologne,	} Thuillier, l. c.
1664	} &c.)	
1670	*France (Guyenne, Sologne, &c.)	Ib.
1672	Germany (Westphalia)	Heusinger, Rech. de pathol. comparée ii, 546 (after Diez).
1674	*France (Montargis)	Dodart, Philos. Transact., 1676, Nr. 130, 758.
„	*Switzerland (Bern, Lucerne, Zurich)	Lange, l. c., ad 1650.
1675	*France (Gatinois) ...	
„	Germany (Westphalia, Voigtland)	Leisner, Hoffmann, ll. cc.
1676	*France	} Lange, l. c.
„	*Switzerland	
„	England	Birch, Philos. Transact.
1687	Germany (Hesse, Westphalia, Cologne)	Act. med. Berol., ii; Ann. vi, 50.
1693	Germany (Black Forest)	Wepfer, Obs. de affect. capitis, obs. 120, Scaph., 1727, 556.
1694	*France (Orleans, Sologne)	Heusinger, l. c. (after Diez).
1699	^o Germany (Harz)	Brunner, Ephem. Acad. Leop. Dec., iii, Ann. ii, Obs. 224.
1700	Germany (Thuringia)	Hoyer, ib., Ann. ix et x, Obs. 93.

Year.	Country.	Authority.
1702	Germany (Hanover, Silesia)	Hoffmann, l. c.; Taube, Gesch. der Kriebelkrankheit, &c., Gött., 1782, 31; Burghart, Med. Siles. Satyræ, iii, 26.
1709	*France (Sologne, Dauphiné)	Dodart, l. c.
"	^o Switzerland (Lucerne)	Lange, l. c.
1710	*France (Orléannais, Dauphiné, Langue-doc)	Noel, Hist. de l'Acad. des Sc., 1710, 80.
"	*Italy (Cremona)	De Renzi, l. c. (after Ginanni).
"	Russia (Baltic Provinces)	Ilmoni, Nord. Sjukd. Hist., iii, 15.
1716-7	Germany (Silesia, Saxony, Lausitz, Sleswig, Holstein)	Bresl. Samml., 1717, 87, 397, Act. med. Berol., ii, Ann. vi, 50; Wedel, Diss. de morbo spasm. epid., Jen., 1717; Waldschmidt, Diss. de morbo epid. convuls., Kil., 1717.
"	Switzerland (general)	Bresl. Samml., 1717, 87.
1722-3	Germany (Silesia, Priegnitz, Pomerania)	Vater, Diss. de morbo spasm. popul., Silesiae, Wittbg.; 1223; Act. med. Berolin, l. c., 54.
"	Russia (Moscow, Novgorod)	Schober, in Bresl. Samml., 1723, 39.
1736-7	Germany (Silesia, Bohemia)	Burghardt, Med. Siles. Satyr., i, 44, iii, 26; Serinci, ib., iv, 35.
1741	Germany (Brandenburg, Westphalia, and other places in North Germany)	Bergen, Diss. de morbo epid. spasm. conv., Feft., 1742; Brückmann, Commenc. litter., Norimb., 1743, hebd. 7; Hofmeyer, Von der Kriebelkr. u. s. w., Berl., 1742; Leidenfrost, Diss. de morbo convuls. epid., &c., Duisb., 1771.
1745	Sweden (Elfsborg) ...	Rosenblad, Act. med. Suec., i, Sect. ii, 9.
1746-7	Sweden (Lund).....	Rosenstein, Diss. de morbo spasm. convuls. epid., Lond., Goth., 1749.
1747	*France (Sologne, Artois, Flanders, Bordeaux)	Du Hamel, Hist. de l'Acad. des Sc., 1748, 528; Salerne, Mém. de l'Acad. des Sc., ii, 155; Raulin, Observ., 320.
1749	*France (Bethune) ...	Tissot, Gesamm. Schriften, v, 687.
"	^o France (Flanders) ...	Bouchet, Journ. de méd., 1762, xvii, 327.
1750	Germany (Potsdam)	Cothenius, in Schreber, Samml. verm. Schriften, i, 413.
1754	Sweden (Småland, Blekinge)	Linné, Amoenitates acad., vi, 430.
1756-7	Germany (Rhenish provinces)	Leidenfrost, l. c., ad 1741.
1764	*France (Artois, Arras, Douay)	Tissot, l. c., Read, l. c., 82.

Year.	Country.	Authority.
1765-6	Sweden (Småland) ...	Wahlin, Abhdl. der Schwed. Akad., xxxiii, 18.
1770-1	Germany (widely diffused, especially in northern districts)	Taube, l. c.; Leidenfrost, l. c.; Wichmann, Beitr. zur Gesch. der Kriebelkr., Lpz., 1771, Nachricht von der Kriebelkr., &c., Zelle, 1771, Bericht, die Kriebelkr. betr. Kopnb., 1772; Steffens, Hannov. Mag., 1771, Nr. 50, 51; Tode, Med.-Chir. Bibl., i, 150; Marcard, Von einer der Kriebelkr. ähnlichen Krampfsucht., Hamb., 1772.
"	*France (Sologne, Maine, Tours, Angou, and other places)	Tissot, l. c., 725; Read, l. c., 83; Vetillart, Mém. sur une espèce de poison, connu sous le nom d'Ergot, &c., Tours, 1770.
1785	Sweden (Blekinge, Kronoberg, Christianstad)	Ref. in Weckoskrift för Läkare, vii, 61, 207, viii, 85.
"	Italy (Tuscany)	De Renzi, l. c., 54 (after Giovanelli).
1785-7	Russia (Kieff)	Frank, Prax. med. univ. praece., ii, vol. i, sect. ii, 205.
1789	Italy (Turin).....	Moscatti, Nuovo giorn. delle più recente Letter med., 1795, ix, 117.
1793	Italy (Foggia, Capitanata)	De Renzi, l. c., 61.
1794	Germany (Stolberg)...	Kortum, Beitr. zur Arzeneiwiss., Gött., 1795, 145.
1795	Italy (Milan).....	Moscatti, l. c.; Corradi, Annal. delle epid. in Italia, iv, 461.
1801	Germany (Treves) ...	Burckhardt, Allgem. med. Annal., 1802, Correspondenzbl., 186.
1804	Russia (Minsk, Pödo-lia, Ukraine, Volhynia, Jekaterinoslav)	Frank, l. c.
1804	Sweden	} Philippoff, Med. Ztg. Russl., 1845, 388.
1805	Poland	
"	Germany (Prussia, Brandenburg)	Lorinser, Vers. und Beobacht., Berl., 1824.
1813-4	*France (Dpt. Saône-Loire, Allier, Isère, Côte-d'Or, Lyon)	Courhaut, Tr. de l'ergot du seigle, Challons sur Seine, 1827; Boucher, Des effets du s. e., Par., 1840; Janson, Mélanges de chirurg., Lyon, 1844, 379; Marmy et Quesnois, Topogr. méd. du Dpt. du Rhône, Lyon, 1866, 184.
1816	*France (Lyon, Dpt. Isère, Drôme)	Compt. rend. de la soc. de méd. de Lyon, 1818, 37; François, Journ. gén. de méd., lviii, 72; Lecompte, Gaz. de Santé, 1817, Jan.; Courhaut, l. c.
1819	Russia (Viatka)	Frank, l. c.

Year.	Country.	Authority.
1821	Germany (Bohemia)	Witterungs- und Krankheits-Constitution in Böhmen, 1821, Prag, 1824.
1824	^o Russia (Dünaburg)	Yassukowitsch, Bull. des sc. méd., xvi, 40.
1825	United States (New York)	Ref. in New York Med. and Phys. Journ., 1825, v, 493.
1829	Germany (Circle of Bunzlav, Bohemia)	Ref. in Oest. med. Jahrb., Nst. F., v, 197.
1831-2	Germany (Circle of Schweinitz, Merseburg, Circle of Luckau, Potsdam)	Wagner, in Hufel. Journ., Bd. 73, Heft 4, 74, Heft 5, 6, 75, Heft 3, Sanitätsber. der Prov., Brandenburg, 1831, 65; Burdach, in Casper's Wochenschr., 1834, 724; Schramm, ib., 1833, 377.
1832	Germany (Braunsdorf, Saxony)	Ref. in Radius' Cholera-Ztg., 1832, Nr. 119, 364.
1835-6	Russia (Novgorod) ...	Bardowski, Med.-Ztg., 1850, Nr. 22, 171.
1837	Russia (around Lake Onega)	Schrenck, Reise in die Tundren der Samojeden, i, 19.
1840-4	Finland (very widely diffused)	Hartman, Finsk. Läk. Sällsk. Handl., i, Nr. 1; Ilmoni, ib., iii, Nr. 1; Spoo, Om förgiftningar med. secale cornutum, &c., Helsingfors, 1872, 23.
1844	Sweden (Kalmar).....	Ekman, Hygiea, 1845, Oct.
"	Russia (Novgorod) ...	Bardowski, l. c.
"	Germany (Circle of Elbogen, Bohemia)	Ref. in Prag. med. Viertelj., 1845, ii, 197.
1845-6	^o Belgium (St. Bernhard, Namur, Ghent)	Bull. de l'Acad. de méd. de Belg., v, 410.
1848	Sweden (Nerike)	Levin, Hygiea.
1851	Norway (Smaalenene)	Ref. in Norsk Mag. for Lægevidensk., 1851, v, 847.
1851-2	Sweden (Kopparberg, Jönköping)	Sveriges Sundh. Berättelse, 1851, 156, 1852, 21.
1852-3	Russia (Govern. Minsk.)	Ref. in Med. Ztg. Russl., 1853, 158.
1854-5	Germany (at various places around Prague)	Hussa, Prag. med. Viertelj., 1846, ii, Analect. 38.
"	*France (Dpts. Isère, Loire, Haute-Loire, Ardèche, Rhone)	Barrier, Gaz. méd. de Lyon, 1855, Nr. 10.
1855-6	*Germany (Brünn) ...	Helm, Woch. der Gesellsch. der Wien. Aerzte, 1856, 165, 186, 197.
"	Germany (Nassau, Brunswick, Waldeck, Hesse, &c.)	Franque, Nass. med. Jahrb., 1856, xiv, 336; Kahler, Correspdzbl. f. Psychiatrie, 1855, Nov.; Pockels,

Year.	Country.	Authority.
1857	Hungary (East of Transylvania)	Deutsch. Klin., 1857, Nr. 1, 2; Heusinger, Studien über den Ergotismus, Marb., 1856, 29.
1862	Finland (widespread)	Meyr, Wochenschr. d. Gesellsch. der Wien, A., 1861, 377.
1867	Germany (especially in Chemnitz)	Spoof, l. c.
1867-8	Sweden (Kronobergs-län)	Flinzer, Viertelj. f. gerichtl. Med., 1868, viii, 360.
1879	Germany (Circle of Frankenberg, Hesse)	Sveriges Sundh. Berättelse, 1867, 33, 1868, 39.
„	Russia (Novgorod) ...	Siemens, Arch. für Psychiatr., 1880, xi, 108.
		Swiatlowski, Petersb. med. Woch., 1880, Nr. 29.

§ 56. TWO FORMS OF ERGOTISM DISTINCT IN TIME AND PLACE.

Without doubt this table of all the epidemics of ergotism that have come to my knowledge represents the *history of the disease* very imperfectly ; and that holds good not only for the mediæval history of the gangrenous disease, but even more for the history of the convulsive form (*Kriebelkrankheit*), of which we have no accounts at all until the sixteenth century. Still, from the accounts that have come down to us, we may draw certain general conclusions as to the history and geography of the disease, which will throw some interesting light on the pathogenesis.

The most noteworthy of all the facts are : that the gangrenous form of sickness and the convulsive (*Kriebelkrankheit*) have at all times had almost entirely distinct areas of distribution, each disease appearing time after time in its own territory as a wide-spread epidemic ; that certain of the greater of these epidemics have coincided in time in the various countries ; and that both forms of ergotism have occurred much more frequently and have been much more widely diffused in former centuries than in recent years.

Of 38 epidemics of gangrenous ergotism recorded between the end of the sixth century and the end of the fifteenth, 22 happened in France,

5 in Spain, 4 in the Netherlands (Flanders), 4 in Germany (3 doubtful), 2 in England (both doubtful), and 1 in Italy. From the beginning of the sixteenth century down to the year 1880, there have been 25 epidemics of ergotismus gangrænosus chronicled, 18 of these belonging to France, 3 to Switzerland, 2 to Spain, and 1 each to Italy and Germany. Apart from the 5 doubtful epidemics, there are thus 58 in all, of which 40 have occurred in France, 7 in Spain, 4 in the Netherlands, 3 in Switzerland, and 2 each in Italy and Germany. As far as the data enable us to decide, there have been thirteen more considerable epidemics of gangrenous ergotism in France—in the years 993-4, 1094-5, 1109, 1128-29, 1151, 1214-15, 1650, 1670, 1709-10, 1747, 1770-71, 1813-14, and 1854-55; in Switzerland there have been two (1650 and 1674), and in Germany one (1486).

Of epidemics of ergotismus convulsivus (*Kriebelkrankheit*) we have accounts of 62 in all between the years 1581 and 1879, of which 29 pertain to Germany, 11 to Russia, 10 to Sweden, 4 to Italy, 2 to Finland, and 1 each to the Netherlands, England, Switzerland, Norway, Hungary, and New York. Of these 62 epidemics, there have been only 8, so far as we can gather, of any considerable extent, namely, 5 in Germany (1595-96, 1716-17, 1741, 1770-71, and 1855-56), 2 in Finland (1840-44 and 1862-63), 1 in Switzerland (1716-17), and 1 in Russia (1804).

The chief coincidences have been the gangrenous epidemics in France and Switzerland in 1650, the convulsive epidemics in Germany and Switzerland in 1716-17, and the gangrenous epidemic of France with the convulsive epidemic of Germany in 1770-71.

§ 57. GEOGRAPHICAL DISTRIBUTION OF ERGOTISM.

Not less noteworthy for the history of ergotism is its comparatively *small area of distribution*, not merely in respect of the surface of the globe generally, but even within the particular regions from which the accounts of it come to us. From *North America*, excepting a small epidemic of the convulsive form limited to a few persons in one of the New York city prisons, I have not been able to find any accounts of ergotism, although I have searched carefully in the very copious medico-topographical and epidemiographical literature of the United States. Nor have I found any certain indication of ergotism in the medical intelligence from *Central America*, the *West Indies*, *South America*, *Africa*, *Asia*, *Australia*, and *Oceania*. The disease would thus appear to be confined exclusively to *Europe*, and within that conti-

ment to certain regions, and within these again to certain definite circles.

Among the European countries that have been subject to ergotism, the first place has to be assigned, according to the data, to *France*, *Germany*, *Russia*, and *Sweden*; from all other parts of Europe, the accounts of epidemics of ergotism are but few.

In *France* the disease (in the gangrenous form) has been observed most frequently in the upper and lower basins of the Loire (provinces of Orleannais, especially in the Sologne, Touraine, Poitou, Anjou, and Maine), and in the basin of the Rhone (provinces of Lyonnais, Dauphiné, Languedoc, and Burgundy). Of forty-seven epidemics whose area is somewhat accurately given, 16 pertained to the first of these regions and 13 to the second; next come 8 epidemics in the south-west (Angoumois and Guyenne), 7 in the north (Lorraine, Picardy, Artois, and Flanders), 2 in the Isle de France, and 1 in Brittany. Of the 19 epidemics which have occurred in more or less wide diffusion over France from the beginning of the seventeenth century to the present time, 9 belong to the seventeenth century, 7 to the eighteenth, and only 3 (of any importance) to the nineteenth. In *Germany* there has been since the sixteenth century only one epidemic of the gangrenous form, a small one in 1855-56 among railway navvies near Brünn; but of *Kriebelkrankheit* there have been 57 epidemics, of which 11 date from the sixteenth century, 10 from the seventeenth, 21 from the eighteenth, and 15 from the nineteenth, the only instance of its general diffusion in recent times having occurred in 1855-56. Of these 57 epidemics, 23 occurred in the north-east of Germany (Prussia, Silesia, Brandenburg, Pomerania, Saxony, and Thuringia), 23 in the north-west (Holstein, Schleswig, Brunswick, Hanover, Westphalia, and Rhenish Prussia), while there were only 8 in the south-west (Hesse, Nassau, and Baden), and only 5 in Bohemia. From the central regions of South Germany (Württemberg, Bavaria, &c.) there is not a single reference to ergotism known. In *Russia*, ergotism in the form of *Kriebelkrankheit* has been especially frequent, having occurred at some places such as Novgorod (according to Bardowski) in the character of an endemic.

The epidemiographical accounts of it from that country for the period between 1710 and 1879 are certainly not more than twelve. It is stated, however, in a paper by Swiatlowski that *Kriebelkrankheit* was prevalent from 1832 to 1864 in the governments of Vladimir, Volhynia, Grodno, Jekaterinoslav, the Don Steppe, Kaluga, Kieff, Minsk, Mohileff, Moscow, Novgorod, Petersburg, Simbirsk, Smolensk, Tauria, Tver, Tomsk, Tula, Charkoff, Tschernigoff, Jaroslav, and more especially in Viatka, Kasan, and Kostroma—so that hardly any part of this vast empire would seem to have escaped. In *Sweden* the first occurrence of *Kriebelkrankheit* appears to have been in 1745; from that date down to 1867 there are ten epidemics of it mentioned in the epidemiographical records of the country, which were all confined to the south excepting the epidemic of 1844 in Nerike (Örebrolän) and that of 1851 in Kopparberg. In *Finland* there were two very severe epidemics in 1840-44 and 1862-63; for *Norway* I find mention of only one small epidemic in 1851.

Ergotism does not appear to have been observed during the present century in any other countries of Europe, excepting *Transylvania* (in the south-eastern portion of which there was a somewhat extensive epidemic of ergotismus convulsivus in 1857), and *Belgium*, where the disease occurred in a few localities to a slight extent in 1845-46. In *Switzerland* the gangrenous form of the disease occurred in the seventeenth century three times (1650, 1674, and 1676), and in the eighteenth century twice in the convulsive form (1709 and 1716). From *England* and the *Netherlands* we have no information whatsoever as to ergotism during the last three centuries. In *Italy* the disease had occurred in former times, but only in occasional and very limited epidemics; Moscati says that it was almost unknown in Northern Italy, and De Renzi¹ observes that the only accounts of it in that country relate to those epidemics which are mentioned by him (and are given in the chronological table). In *Spain* also it does not appear that the malady has recurred during the last three centuries.

¹ 'Storia della medicina in Italia,' Napol., 1848, v, 763.

§ 58. CONDITIONS FAVORABLE TO THE GROWTH OF ERGOT ON RYE.

There is no doubt that *ergotism is due to the somewhat free use of corn contaminated with the ergot-parasite*, that is to say, of articles of food prepared from the corn ; and it appears to be equally certain, that although the same parasite grows upon various other graminaceous plants, yet it is only when it occurs on rye, and perhaps also on the bromus-grass,¹ that it possesses the poisonous property. We find evidence of this, apart from experiment, in the fact that the disease has occurred most frequently, and to the extent of an epidemic, under circumstances which are known from experience to be most favorable to the thriving of the corn-parasite, as, for example, after a *damp season*, particularly in years where a hot and dry summer has followed a very rainy spring, or where the corn has grown in the shade, or in localities where the bottom is sandy, cold² or marshy, or where the grain is ill-formed ; and we shall probably not err if we seek to find the reason of the comparatively rare occurrence of ergotism in recent times in the more careful cultivation of the soil, in the improved food-supply due to better ways and means of communication, and in the general cultivation of the potato.

The influence of the above-mentioned weather-conditions on the production of the disease, or, in other words, on the morbid causation, has been apparent in a very large number of epidemics. Facts pointing that way were observed in Flanders in 1094-95, France and Switzerland in 1650, 1674, and 1676, France in 1710, Saxony and the Lausitz in 1716, Pomerania, Silesia, and the Priegnitz in 1723, Silesia and Bohemia in 1735-37, Brandenburg and Westphalia in 1741, Sweden in 1745, 1754, 1765, and 1785, Lille in 1749, North Germany in 1770-71, the circles of Schweinitz and Luckau in 1831-32, Finland in 1840-41, Belgium in 1845-46, the north-west of Germany in 1855-56, and Transylvania in 1857. The significance of a marshy soil for these occurrences comes out very definitely in the almost endemic prevalence of the disease on the marshy banks of the Loire, particularly in the Sologne and Blaisois. Another noteworthy fact, observed in 1749 in

¹ Heusinger makes out a particularly good case for this as regards the epidemic of 1855-6 in Upper Hesse.

² Observations to that effect were made in 1770 in Schleswig and Lüneburg, in 1840 in Finland, and in 1844 in Sweden

the neighbourhood of Lille, is that the disease was much more widely spread on the marshy ground to the south of the town than on the dry ground on its northern side. The influence of the cultivation of the soil on the occurrence of ergot is shown in the fact given by Riva,¹ that since a principle of rotation was introduced into the rice fields of Northern Italy, the fields being worked at one time under irrigation and another time dry, the crop of rye grown on the fields that had been watered abounds in ergot.

§ 59. MOSTLY A DISEASE OF THE POORER PEASANTRY AND OF CHILDREN IN PARTICULAR.

However sound may be the general conclusions arrived at as to the origin of ergotism, there still remain some problems in the history of this disease, the solution of which is for the present not entirely satisfactory.

The first noteworthy thing is that ergotism has occurred almost exclusively among the *country population*, so much so that some practitioners have thought themselves justified in calling the disease "morbus ruralis." Next we have a fact ascertained in all epidemics of ergotism, that the disease has been prevalent mostly if not exclusively among the *poorer class of people*, and has attained its widest distribution when prices have been high owing to failure of crops or to other calamities, or when there has been actual famine.² In the same connexion we may take the fact sometimes observed, that ergotism may confine itself exclusively to places *exceptionally ill off in hygienic respects*—to prisons, foundling hospitals, orphanages, and the like. Instances have occurred at Turin in 1789 and Milan in 1795, both times in orphanages (Moscatti), in the prison of Treves in 1801-2, in a prison at New York in 1825, in a children's institute at Bräunsdorf (Saxony) in 1832, and in the prisons of St. Bernard, Namur, and Ghent in 1845-46. Finally, it has happened not unfrequently that the age of *childhood* has furnished the largest contingent of the sick, as in Stolberg in 1794,

¹ 'Influenza delle rizaje,' Novara, 1847, p. 35.

² Examples of this are furnished by the epidemics of 1581 in Lüneburg, 1695 in the Harz, 1741 and 1770-1 in North Germany and in France, 1804-5 in Sweden and Poland, 1831-2 in the circle of Schweinitz, 1835 and following years in Novgorod, 1848 in Nerike (Sweden), and 1854-5 in several parts of France.

in the circle of Luckau in 1831-32, Sweden in 1754, Beaurepaire (Dauphiné) in 1816, Upper Hesse in 1855-56, and in the epidemics already mentioned as occurring at Turin, Milan, and Bräunsdorf in 1789, 1795, and 1832. All these facts are to be explained, in my view, by the influence which unfavorable hygiene and diet exert upon the individual, inasmuch as they lower his power of resistance to noxious things, and thereby increase his predisposition to fall into sickness; and for the remarkably frequent incidence of the disease in childhood a corresponding reason may be found in the well-known liability of that particular time of life to be affected by narcotic poisons.

But it is not so easy to explain how it is that poisoning by ergot has led to gangrenous disease in some regions (France and Spain), and in other regions (Germany and Russia) just as uniformly to convulsive ergotism (*Kriebelkrankheit*); and why it has happened that only on the rarest occasions the two forms of disease have occurred together. It is highly improbable that this variation in the type of the disease depends on the quantity of poison consumed; it is much more reasonable to conjecture that we have here to do with a qualitative difference in the toxic principle of the ergot parasite, a difference which would appear to be essentially bound up with the soil on which the epiphyte thrives.

CHAPTER VI.

PELLAGRA AND ALLIED DISEASES.

§ 60. SYMPTOMS AND MORBID ANATOMY OF PELLAGRA.

Under the name of “pellagra,” corresponding to the Spanish *mal de la rosa* or *mal roxo*, a peculiar disease has come to knowledge, very distinctively characterised by a series of morbid conditions in the skin and the mucous membrane of the digestive tract, and by symptoms referable to the cerebro-spinal system. It showed itself first about the middle of last century in several parts of Spain and Upper Italy; it came afterwards to Central Italy, and since the beginning of the present century it has broken out in some of the south-western departments of France, and more recently in Roumania and Corfu. A prominent place among the endemic maladies of those regions has been accorded to it, not only on account of its considerable diffusion at many places therein, but also by reason of its injurious influence on the working power of the population.

Clinical history.—The onset of the disease¹ is usually denoted by a sense of weakness and disinclination for the individual's ordinary avocation. He complains at the same time of headache, giddiness, singing in the ears, and an acute feeling of burning issuing from the back, spreading over the extremities and locating itself particularly in the hands and feet. The tongue is coated, the epigastric region, and sometimes the lower part of the abdomen also, are tense and painful, and the stools not unfrequently loose. These phenomena occur in most cases in the beginning of spring; and along with them there breaks out, on those parts of the body which are ordinarily bare and exposed to the sun (the back of the hands, the face, also the feet in those who go bare-

¹ The writings quoted in the sequel are given at the end of the chapter, arranged alphabetically according to the authors' names.

footed, and more rarely the back and chest), an exanthem of irregular formation, bright red or dark red, and sometimes even livid, having the type of erythema or erysipelas, and vanishing completely on pressure. The skin looks to be somewhat swollen at the affected places, and the patient complains of a feeling of tightness, uneasiness or burning, which becomes particularly acute under the action of the sun's rays. After lasting three or four months, that is to say, until July or August, these symptoms decline, the skin remaining somewhat dark-coloured and remarkably rough and dry for a certain time longer; and therewith the morbid process appears to have come to an end. Next spring, however, the whole series of phenomena recurs, and now, or it may be not until the third onset, the disease assumes a more severe character. The general feeling of weakness is now so great that the patient keeps on his feet with difficulty, being unable, accordingly, to pursue his occupation; he loses weight, the pains in the head and back become very acute, and drooping of the upper eyelid, dilatation of the pupil, amblyopia, diplopia, and other disorders of vision follow. (In 50 patients with pellagra, the ophthalmoscopic examination of the fundus of the eye showed the condition to be normal in 10 only, in 29 there was cloudiness or greyish discoloration of the retina, in 23 there was atrophy of the arterial retinal vessels, in 2 there was dilatation of the retinal veins, in 4 atrophy of the papilla, and in 3 redness of it.¹) The exanthem now spreads over larger areas and looks darker coloured, the skin thickens and cracks, the tongue becomes red and dry (the patient complaining of burning in the mouth and pain on swallowing), the coated gums bleed readily, the diarrhœa increases, and most of all the symptoms referable to the cerebro-spinal affection show a marked aggravation. Besides pains in the head and back, tonic and clonic spasms occur in the upper and lower extremities, being sometimes confined to one side; there is also delirium, or a succession of typhoid symptoms under which the patient quickly succumbs. In another and commoner class of cases, a mental disorder gradually sets in, less frequently in the form of mania than of melancholia, with a peculiar tendency to suicide, especially by drowning (hydromania). Other noteworthy signs are: that the extensors get into a state of paresis,² so that the limbs are in the position of semi-flexion owing to the preponderance of the flexors, and remain fixed when an attempt is made to move them; that the electro-muscular irritability of the extensors is diminished; and that there is anæsthesia or lowering of the cutaneous sensibility under the application of the induced current. As the disease advances, it may sometimes be observed that the area of cardiac dulness is diminished, the cardiac impulse feeble, and the heart-sounds less audible. In severe cases the urine is often alkaline, and the specific gravity reduced, it may be as low as 1005; but albumen

¹ Lombroso (ii, 65), after the researches of Flarer, Manfredi, and Forlanini.

² Accurate inquiries into these motor and sensory disorders were first made by Lombroso; they are given at p. 71 of the second of his works quoted in the list at the end of the chapter.

is rarely present. The downward progress of the patient continues, the extremities and the bladder become paralysed, mental power is lost even to the degree of imbecility, and death—if it have not been anticipated by suicide—is ushered in by rapid wasting, colliquative and ill-smelling sweats, profuse diarrhœa and dropsy. A favorable issue of the disease is only to be looked for if there had been no more than one preceding attack, or attacks of the slighter kind if more than one, and the patient then placed under better conditions of living or withdrawn from the morbid influences. When the malady has reached a more advanced stage of development, the prognosis is very gloomy; and even in the best cases there will always be some disorders remaining behind, especially in the sphere of the nervous system. The duration of the disease is exceedingly variable; it may extend to ten or fifteen years or more, and even when it has lasted as long as that, it may still be short of its full development.

Morbid anatomy.—The following are the most noteworthy points in the morbid anatomy as compiled by Lombroso¹ from 66 necropsies made by himself. In 29 cases the pia mater and arachnoid were opaque and thickened (purulent deposit in 4, and ecchymoses under the arachnoid in 5), in 24 cases there was œdema of the brain, in 11 atrophy of the brain, especially the cerebral cortex, in 33 rottenness of the heart-muscle and yellowish-brown discoloration, in 19 (out of 26 examined) the heart under weight, in 16 brown atrophy of the liver, in 40 the spleen atrophied, in 21 fatty degeneration of the kidneys, and in 31 cirrhotic atrophy of the same. There was also that attenuation of the muscular coat of the intestine which all previous observers had noted, with hyperæmia of the mucous membrane and ulceration of the rectum; in 18 (out of 42 where it was looked for) there was fragility of the ribs, the other flat bones being normal, and the individuals not in a state of marasmus nor aged, and in 21 (out of 44 examined) the voluntary muscles were atrophied. From the microscopic examination of the organs most implicated, which was carried on with the assistance of Professor Bizzozero, the following were the chief conclusions: of 33 hearts, brown atrophy of the muscle in 27 (always with splitting of the fibres or formation of lacunæ in them), and fatty degeneration in 5; of kidneys from 28 cases, fatty degeneration of the tubular epithelium and sclerosis of connective tissue in 18; of 27 livers, fatty infiltration of the acini in 12; in 10 brains, pigmentation or fatty degeneration of the outer coat of the capillaries, and in 3 brains calcareous deposit in the same; deep pigmentation of the sympathetic ganglion-cells in 8 cases out of 12. According to the *post-mortem examinations*, the anatomical changes characteristic of the morbid process may be referred to four classes: (1) hyperæmias and inflammatory processes leading to exudation, hypertrophy and the like, as shown in the brain-membranes, the liver, the spleen, the kidneys

¹ L. c., p. 117.

and the lower part of the intestinal canal; (2) atrophy and marasmus especially apt to occur in the heart, lungs, liver, spleen, intestine and kidneys, these being the thoracic and abdominal organs supplied by the vagus and sympathetic nerves; (3) fatty degeneration; and (4) pigmentary changes—a special characteristic of the morbid process.

Lombroso's¹ inference from clinical observations and the post-mortem condition is that pellagra is *primarily a toxic effect on the sympathetic nervous system*, the structural and functional disorders being dependent thereon. Dejerine, who has lately investigated the morbid changes in the cutaneous nerves in pellagra, regards the skin affection as also "d'origine trophique."

§ 61. HISTORY AND PRESENT DISTRIBUTION.

Spain.—The earliest information about pellagra comes from *Spain*. In that country, according to Casal,² it had been observed as an endemic since 1735 in the Asturian district of Oviedo, comprising about a twentieth part of the whole province, while it was quite unknown throughout all other parts of Asturias, including Santillana in the immediate neighbourhood, as well as in the frontier districts of Galicia and Leon. Concerning the later history of this disease in Spain, the facts known to me are very incomplete and not altogether trustworthy; at all events the subsequent diffusion of the malady has been somewhat extensive, especially in the northern provinces of the country. At the instigation of the Italian consul-general in Barcelona, the Academy of Medicine of that city instituted in 1879 a minute inquiry into the present condition of the disease in Spain, the following being the conclusions³ (which may be accepted in part only): *Asturias* is to the present day one of the chief centres of the *mal de la rosa*, the disease being located especially in the communes of Regueras, Llanera, Corbera, and Careño. It is met with also, to no inconsiderable extent, in

¹ L. c., p. 97.

² Casal's treatise was first printed in 1762, although its contents were known before to Thiéry, who had brought the description of the *mal de la rosa* under the notice of the profession in France ('Journal de méd.,' 1755, ii, 337).

³ This report is given in the official publication of the Italian Government, 'La Pellagra,' Roma, 1880, p. 387.

Lower Arragon and *Burgos*, as well as in the province of *Guadalajara*, where it is so prevalent in several of the fifty villages between *Colmenar di Oseja* and *Vasouaña* on the one side and between *Sadices* and *Brihuega* on the other, that the number of the sick may amount to 2 per cent. of the population. Information to the same effect comes from several parts of the province of *Cuenca*, from the district on the frontiers of *Navarra* and *Arragon* (*Sanguesa*), from the *Ebro* valley in the province of *Zaragoza*, from the level banks of the *Douro* and *Tormes* in the province of *Zamora*, and from *Galicia*. That the disease occurs in many other parts of Spain is shown by the statistics of pellagrous persons received into the hospital of Madrid; of these 10 came from the Madrid province, 5 from Toledo, 3 from Seville, 2 from Lugo, and one each from the provinces of Granada, Orense, Oviedo, Ciudad Real, Guipuzona, Avila, Palencia, Guadalajara, Valladolid, and Segovia. According to the same report, there has been a considerable abatement of recent years, if not in the number of patients, yet in the severity of type of the disease.

Italy.—Shortly after it began in Spain, pellagra made its appearance in *Italy*—in *Lombardy* and *Venetia*. Even previous to 1730, occasional cases had been seen¹ in the vicinity of *Sesto Calende* (on *Lago Maggiore*); but from that date the cases became more numerous, the malady breaking out simultaneously in the districts of *Milan*,² *Brescia*,³ *Bergamo*,⁴ and *Lodi*,⁵ and shortly after in country around *Como*,⁶ *Cremona*,⁷ *Mantua*,⁸ and *Pavia*; ⁹ so that towards the end of the century its area extended over nearly the whole of *Lombardy*.¹⁰ In *Venetian* territory, it showed itself first at *Udine*,¹¹ and we have information of it a little later in date from the provinces of *Belluno*,¹² and *Padua*,¹³ and then from *Verona*; ¹⁴ so that the *Venetian* kingdom was also affected by it over a

¹ Terzaghi.

² Frapolli, Zanetti, Gherardini, Albera, Strambio.

³ Balardini, Menis, Mottoni, 'Relazione.'

⁴ Villa.

⁵ Cerioli, Tassani, I, Robolotti, Cappi.

⁶ Hildenbrand, Cambieri.

⁷ Pujati, Romano.

⁸ Fanzago.

⁹ Facheris, Chiappa.

¹⁰ Comolli, Tassani, II.

¹¹ Sacchi, Lombroso, I.

¹² Jansen, Cerri, Balardini.

¹³ Odoardi, Zecchinelli, Pertile.

¹⁴ Agostini.

wide area before the end of last century or the beginning of the present.¹ In *Piedmont* and *Liguria* it is of more recent origin; our first information of its occurrence in them dates from the last ten years of the eighteenth century, when it was seen at Castellalfero² and in the territory of Massa;³ since 1820-30, it has attained to general diffusion in these regions, especially in the provinces of Ivrea, Saluzzo, and Alessandria, but nowhere to the same extent as in Venetia and Lombardy.⁴ The Genoese littoral has continued free from it down to the present day.⁵ In *Tuscany* it was known as early as 1785, it showed itself afterwards in 1797-98 around Mugello, reappearing there to a greater extent in 1809, and in the Romagna Toscana;⁶ in more recent years it has established endemic centres in the upper valley of the Arno, in Volterrano, around Lucca and Pisa, and among the hills near Florence.⁷ The same period at which the disease was first seen in Tuscany furnishes us with the earliest accounts of its occurrence in the *Æmia*; we hear of it at Reggio⁸ in 1782, in the vicinity of Bologna⁹ and in the district of Parma¹⁰ at the beginning of the century, while its appearance in Ferrara¹¹ and Modena¹² was somewhat later, and its more considerable outbreak not until recent times. The same applies also to the development of the disease in the *Marches* (mostly in the province of Urbino-Pesaro),¹³ and in *Umbria*;¹⁴ but in these, as well as around Rome,¹⁵ it has never attained the same importance as in Northern Italy. The southern provinces of Italy (the Abruzzi, Campania, Apulia, Basilicata, and Calabria), as well as Sicily and Sardinia, have hitherto enjoyed immunity from the disease.

¹ Soler, Facen, Festler, Liberali.

² De Rolandis, I.

³ Boërio.

⁴ Fontana, De Rolandis, II, Ferraris, Ramati, Garbiglietti, Maffoni, Girin, Vacca, I.

⁵ Morelli.

⁶ Vignoli.

⁷ Chiarugi, Cipriani, Vignoli, Morelli, Bartolozzi.

⁸ (Rosa.)

⁹ Calori, Farini, Paolini, Leonardi, Brugnoli.

¹⁰ Guerreschi, Thomasini.

¹¹ Gambieri, Bennati.

¹² Martinelli, Maragliano, Vacca, II.

¹³ Girolami, Frigerio, Michetti.

¹⁴ Adriani.

¹⁵ Farini.

The following table gives the amount of pellagra in the several divisions and provinces of Italy in 1879:¹

Divisions and provinces.	Population.		Number of cases of pellagra.	Proportion of cases of pellagra per 1000 inhabitants.	
	Urban and rural.	Rural alone.		Urban and rural.	Rural alone.
LOMBARDY	3,653,941	1,284,670	40,838	11·2	31·70
Pavia	477,887	172,758	800	1·7	4·63
Milan	1,070,998	322,320	10,380	9·8	32·20
Como	513,677	161,964	618	1·2	3·81
Sondrio	118,835	57,274	39	0·3	0·68
Bergamo	389,406	153,418	7,122	18·3	46·42
Brescia	468,906	187,278	14,989	31·7	80·03
Cremona	313,143	116,728	5,235	16·7	44·84
Mantua	301,089	112,930	1,655	5·5	14·65
VENETIA	2,812,022	977,346	29,386	10·5	30·52
Verona	388,489	125,722	2,391	6·2	19·01
Vicenza	393,250	146,788	3,400	8·1	23·16
Belluno	190,491	66,090	1,400	7·4	21·18
Udine	509,447	189,054	4,000	7·9	21·15
Treviso	382,410	152,186	4,902	12·8	32·21
Venice	346,851	77,878	2,696	7·8	34·61
Padua	386,762	143,024	8,207	21·2	57·38
Rovigo	214,322	76,604	2,840	13·3	37·07
PIEDMONT	3,077,200	1,147,808	1,692	0·6	1·47
Cuneo	653,903	277,886	34	0·05	0·12
Turin	1,021,630	355,688	1,042	1·0	2·93
Alessandria	728,941	308,570	403	0·6	1·31
Novara	672,726	205,664	213	0·3	1·03
LIGURIA	1,056,669	310,552	148	0·14	0·47
Porto Maurizio	130,000	54,904	—	—	—
Genoa	755,428	193,166	27	0·004	0·14
Massa and Carrara...	171,241	62,482	121	0·07	1·93
EMILIA	2,193,440	791,408	18,728	8·53	23·66
Piacenza	230,713	83,968	4,326	18·75	51·51
Parma	270,456	109,436	5,013	18·53	45·80
Reggio	250,570	95,564	920	3·68	9·62
Modena	281,593	100,158	1,500	5·33	14·97
Ferrara	228,931	65,946	3,650	15·43	55·34
Bologna	455,190	157,846	2,574	5·63	16·30
Ravenna	229,866	83,836	145	0·64	1·72
Forli	246,121	94,654	600	2·45	6·33

¹ I have taken the figures from the official publication 'La Pellagra in Italia,' Roma, 1880. The calculation of the number of cases in proportion to the urban and rural population has a special interest; for it shows us that pellagra is met with almost exclusively in the latter class of the population, while, in contrast, it is absent among the inhabitants of the towns.

Divisions and provinces.	Population.		Number of cases of pellagra.	Proportion of cases of pellagra per 1000 inhabitants.	
	Urban and rural.	Rural alone.		Urban and rural.	Rural alone.
TUSCANY	2,048,537	691,694	4,382	2.17	6.33
Lucca	292,651	100,660	2,500	8.56	24.83
Pisa	280,406	90,312	22	0.08	0.24
Leghorn	120,000	9,908	—	—	—
Florence	796,447	228,494	560	0.71	2.45
Arezzo	239,033	118,042	1,300	5.86	11.01
Siena	210,000	95,248	—	—	—
Grosseto	110,000	49,030	—	—	—
MARCHES AND UMBRIA	1,498,284	619,534	2,155	1.44	3.47
Pesaro	222,765	91,688	1,000	4.49	10.90
Ancona	271,397	88,092	300	1.10	3.40
Macerata	244,646	103,774	225	0.09	2.17
Ascoli Piceno	209,476	85,798	40	0.02	0.46
Perugia	550,000	250,182	590	1.08	2.36
ROME	849,125	581,939	76	0.09	0.25

According to this table the territories that suffer most are Lombardy, Venetia, and the *Æmilia*: in Lombardy chiefly the provinces of Brescia, Bergamo, Cremona, and Milan; in Venetia the provinces of Padua and Rovigo; and in the *Æmilia* the provinces of Ferrara, Piacenza, and Parma. When we follow the distribution of the disease into the several districts of these provinces, we find the largest number of cases in the district of Verolanuova (Brescia), being 59.6 per 1000 of the whole population; and next in order in Badia (prov. Rovigo) with 54.6 per 1000, Conselve (Padua) with 50.2, Chiari (Brescia) and Campo San Piero (Padua) with 42.9, Borgo San Domino (Parma) with 31.7, Brescia district with 31.2, Lodi (prov. of Milan), and Treviglio (Bergamo) with 27.5, Asiago (Vicenza) with 24.7, Este (Padua) with 23.5, Cento (Ferrara) 23.0, and the districts of Cremona and Piacenza with 22.5 per 1000.

Another noteworthy fact is that, in proportion as the area of pellagra has extended into Central Italy, the number of cases in the earlier seats of the disease has increased.¹ Thus, to mention only a few instances, the number of

¹ The only considerable exception to this rule is the province of Cuneo (Piedmont), where the number of pellagra cases was 294 in 1847, but only 34 in 1879.

pellagrous persons in Lombardy in 1839 was 20,282; in 1856 it had risen to 38,777, and in 1879 it had reached the figure of 40,838. In Piedmont, an enumeration in 1847 showed the number to be 987, while in 1879 it was 1692. In Venetia, 20,000 cases of pellagra were known between 1853 and 1856, whereas in 1879 the number was 29,386. As an instance of the way in which this increase has affected the several provinces, we may take the case of the province of Vicenza, where there were only 1380 pellagrous subjects in 1853-55, but 2974 in 1860, and 3400 in 1879.

France.—Besides those of Spain and Italy, the endemic seats¹ of pellagra are a few districts in the south-west of France, as well as Roumania and the island of Corfu. The first intelligence of pellagra in France² dates from 1829, in which year Hameau published an account of cases observed by himself since 1818 around Teste-de-Buche and in the plain of Arcachon, as well as of the subsequent increase and general diffusion of the malady in the coast-region of the *Gironde*. His facts were afterwards confirmed by Gintrac; but this observer, as well as others,³ brought forward evidence at the same time that the endemic focus of pellagra was by no means confined to that part of Gascony which lies between the left bank of the Garonne, its tributary the Ciron and the coast, but that it covered a large area in the department of *Landes* as well, its extent being indeed greater in the latter than in the former.⁴ Smaller centres of pellagra, of more recent origin, are met with in the departments of *Hautes-Pyrénées*

¹ The statements as to the occurrence of pellagra in Hungary and the Banat, in Egypt, and in Algiers, are based upon quite untrustworthy information. Pruner's description (l. c., p. 179) of the cases which he saw in Egypt (a brownish exanthem, paresis of the upper extremities, and muscular atrophy), does not by any means correspond to pellagra; and as regards De Bucherie's account ('*De la Pellagre, &c.*' Strasb., 1858), in which mention is made of the disease occurring in Constantine, we have the testimony of both Armand ('*Méd. et hyg. des pays chauds*,' Paris, p. 428) and Bertherand (as quoted by Pietra Santa, in '*Journ. d'hyg.*' 1880, Dec., p. 619) that pellagra has never been seen in Algiers, unless a very doubtful case of Armand's be regarded as such.

² Roussel gives a complete history of the disease in France down to the year 1845.

³ Marchand, Hiard, Sorbets, Balhadère, Bouchard, Lavielle, Lalesque.

⁴ In the arrondissement of Dax (Dept. Landes) the disease, according to Lavielle, has decreased of late.

and *Basses-Pyrénées*,¹ in the valley of Vernet (*Pyrénées orient.*),² and in the plain of Lauragais—in the arrondissements of Villefranche (*Haut-Garonne*)³ and Castlenaudary (*Aude*)⁴. At all these points, however, and especially at the last named, the amount of the disease is very little. Observations on sporadic cases of pellagra come to us from some other parts of France—from *Paris*,⁵ the departments of *Seine-Oise*,⁶ *Marne*,⁷ *Allier*,⁸ *Maine-Loire*,⁹ *Ille-et-Vilaine*,¹⁰ and from *Rouen*.¹¹ I shall specially recur to these when I come to speak of the etiology of the disease.

Roumania.—Caillat, who was the first to mention the occurrence of the disease in Roumania, says that it had not been seen there previous to 1846, a statement which v. Theodori confirms, to this extent at least, that his father, who acted as physician to the first hospital in Roumania, assigned the outbreak of the malady to the year 1833, although it was not until 1846 that the first pellagra patients were admitted into the hospital. At the present day, the disease is somewhat widely spread throughout that country, more in Moldavia than in Wallachia,¹² in which latter Felix saw the first cases in 1859-61 at the village of Muscel in the district of Campulungu. The attention of the profession was first drawn to the occurrence of pellagra in Roumania by the work of v. Theodori; since then there have been numerous writings on the disease in that country,¹³ from which it appears that its head-quarters are the districts of Ott (Slatina), Arges (Pitesti), Dimbovitza, Prahova, Buzen, Neamtzu (Piatra), Succava (Folticeni), and Dorohoi. We learn from Felix that the disease since 1878 has declined in some districts and increased in others; he estimates the number of pellagrous persons in Roumania at 4500, or not quite 1 per 1000 of the whole population.

Corfu.—In *Corfu*, according to Typaldos, the first cases of pellagra were seen in 1839, but it is only since 1856 that the disease has taken on the character of an endemic. At

¹ Bataille, Nogués, Laurens, Balhadère.

² Courtz.

³ Calés.

⁴ Roussilhe.

⁵ Roussel (II), Villemin.

⁶ Gibert.

⁷ Landouzy, Collard.

⁸ Bougière.

⁹ Fabre, Billod.

¹⁰ Id.

¹¹ Leudet.

¹² See Champouillon, Scheiber, Klein, Felix.

¹³ A list of all the papers on pellagra in Roumania (most of them in the Roumanian language) is given by Felix (pp. 27-29).

the date of his writing (1867) it was prevalent in 27 out of the 117 rural communes in the island, but only to a moderate extent, the number of cases representing about 3·2 per 1000 of the population.

§ 62. A RECENT DISEASE OF THE PEASANTRY, LIMITED TO A FEW REGIONS.

There are two points in the history of pellagra which give the inquiry into *the origin of the malady* a definite direction from the outset. One is the fact that pellagra has appeared within comparatively recent times as a disease hitherto unknown; the other is that it is limited to an exceedingly narrow area. As regards the first point, we have not only the unanimous opinion of those who observed and have described the first outbreak of the malady in Spain, Italy, France, Roumania, and Corfu; but we have also the circumstance that in the earlier medical records of the countries which subsequently became and continue to be affected with pellagra—particularly in the medico-topographical accounts by Italian physicians of the seventeenth century who gave special attention to prevailing diseases, and above all in the classical work of Ramazzini on the diseases of the labouring class—we do not find the slightest reference which could be taken as bearing even remotely on pellagra.¹ In respect to the second point it is noteworthy that the localities where the disease is endemic are situated within a zone extending from 42° to 46° N., and that within these narrow limits of distribution, it is always and everywhere among the *rural population only* that the endemic foci of the disease exist, the cases of pellagra that come to notice among the residents in towns being at no place or at no time more than sporadic.

“Su tutti i diversi punti di lontanissime parti terraquee,” Lussana and Frua² observe by way of summary, “nelle Asturie spagnuole, nelle

¹ Mottoni points out that in the very carefully kept death-registers of the towns of Chiari and Rudiano, which were among the most intense centres of pellagra shortly after it broke out, there is not a single death entered during the sixty years preceding that outbreak, in which the description of the cause of death points even remotely to pellagra.

² (II), l. c., p. 67.

Lande francesi, nell' Insubria lombarda, sul Veneto, nel Piemonte, nella media Italia—le prime e consuete vittime della pellagra si mostrarono sempre gli abitatori delle campagne e più propriamente i coltivatori dei campi.” Frank¹ says: “Tria solum numeramus pellagrae in incolis urbium exempla;” and he calls attention to the remark of Cerri that many of the cases of pellagra seen in towns are in country people who have migrated thither with the disease already on them. Another telling piece of evidence for the prevalence of the disease among the rural population is furnished by the statistical returns of sickness and mortality.² Of 1955 persons who died of pellagra in Lombardy from 1848 to 1859, the number of country people was 1853 (Lombroso); the whole of the 150 pellagrous lunatics in the asylum at Modena were from the country; among 148 insane patients with pellagra, Salerio found only 9 who were not peasants, and even these were mostly born of country parents; of 561 pellagra-lunatics treated by Vigna, 493 were villagers.³ The proportion has been found to be the same in the more recent outbreak of the disease in Corfu (according to Typaldos) and in Roumania (according to Klein).

“Le petit nombre de cas de vraie Pellagre,” says Felix with reference to Roumania “observés dans quelques villes, s’explique par le fait, que les habitants des quartiers excentriques de ces villes sont des agriculteurs qui vivent dans les mêmes conditions que les paysans.”

These facts, then, warrant us in concluding as regards the source and character of the *actual and material disease-agent*, that its origin is somewhat recent; that it must have developed from time to time in the several pellagrous regions out of causes acting locally; and, accordingly, that all those noxious influences arising from general conditions of *climate*, *weather*, and *soil*—influences which have been felt not only at all times in the localities in question, but have been equally active, and are still as active, at other points of the globe that are exempt—can be concerned in the production of pellagra only in so far as the development of the specific cause of the disease is more or less dependent upon them. This is a point to which I shall return.

¹ L. c., p. 284.

² See the above-quoted statistics of pellagra for the Italian provinces subject to the malady, in which the proportions of sickness among the combined urban and rural population and the rural alone are given side by side.

³ See Parola, ‘Saggio di climatologia e di geographia nosologica dell’ Italia,’ Torino, 1881, p. 754-5.

§ 63. NOT DISTINCTIVELY CAUSED BY POOR LIVING.

These are factors in the etiology whose influence is merely an indirect one ; and along with them I feel bound to include the noxious effects of *wretched living*, particularly the much-blamed inadequacy of the food to the severe labour, the miserable dwellings, and the other things associated with penury. I feel bound to consider those factors also as indirect ; although, as we shall see, there is another way of interpreting the fact (to which all observers bear witness), that pellagra is *associated almost exclusively with the poorest of the rural population*, as well as the other fact attested by many that amelioration in the manner of living, and particularly an improved diet, exerts a favorable influence upon the sick. At all events the opinion held by many observers¹ that pellagra is the direct consequence of poverty, misery, and deficient food, or that it is a "morbus miseriaræ," must be decidedly rejected as untenable.

The misery of living is as old as the human race, and it is no greater in Northern or Central Italy, in the northern provinces of Spain, or in the south-western departments of France, than in many other parts of these countries, or than in Russia, Ireland, Upper Silesia, and Galicia, which have never been afflicted with pellagra. The outbreaks of the disease in Spain, France, Roumania and Corfu were preceded by no noticeable social change of any kind, such as would have caused an increase in the difficulties of living within the affected districts, and so given rise to the development of the disease. Still less can this be alleged of Northern Italy where, about the middle of last century or at the very time when pellagra was beginning to show itself, the social condition of the agricultural community was exceedingly good, while the populations of the States of the Church, who escaped the disease, were groaning under all the stress of misgovernment. Objection had been already taken by Zecchinelli

¹ Menis (I, 138), Mugna, Verga, Gemma, for Lombardy ; de Rolandis, Maffoni, for Piedmont ; Morelli, for Tuscany ; Bennati, for Ferrara ; Cazenave, Courty (l. c., 696), Marchand ('Docum.,' 214), Gintrac (II), Bouchard, Monribot (l. c., 25), Laurens, for France ; Scheiber, for Roumania.

and other of the earliest observers of the disease to the notion that want and misery were essential causes of pellagra, and the same view was afterwards expressed by Frank¹ and others. In the year 1830, when the disease was widely diffused through the provinces of Brescia, Cremona, and Bergamo, there were only two pellagrous persons known in the Val Tellina, “sebbene grande e non minore al certo che altrove vi sia la miseria e il mal nutrimento nei contadini” as Balardini adds;² and in the year 1879 the official returns gave the number of pellagra cases in the whole of Sondrio, a province not specially favoured by nature, at thirty-nine only, while in the other provinces of Lombardy the cases were reckoned by thousands.

§ 64. EVIDENCE THAT IT IS DUE TO DAMAGED MAIZE.

The specific noxious thing on which pellagra depends must consist in some factor which is common property to the affected districts only, and which did not begin to make itself felt until the period from whence our first information about pellagra comes. Starting from these perfectly reasonable premisses, the earliest observers of the disease in Spain and Italy, as well as those of a later date in France, adopted the opinion that the production of the malady was directly caused by the *use of maize as food*; and at the present day that is the doctrine, based on a large experience and on many experiments, which is held, if not without dissent, yet by the great majority of observers and investigators, among whom there are differences of opinion only in so far as relate to the conditions under which a diet of maize becomes a cause of the disease. I accept this theory in the sense of those who take *pellagra to be a toxic process, resembling ergotism, and caused by a morbid condition of maize*; and in the paragraphs that follow I have put together the arguments which have been adduced, or which can be adduced, for and against that view.

(1) Pellagra is *endemic* only in countries where maize is

¹ L. c., p. 289.

² ‘Topogr. statist.-med. della provincia di Sondrio,’ Milano, 1834, p. 65.

cultivated, and where it constitutes a chief article of diet for the mass of the peasantry, particularly in the form of porridge—the polenta of the Italians, the cruchade of the Gascons, and the mamaliga of the Roumanians. On the other hand, in districts lying within the pellagra-areas or directly adjoining them, in which maize, in the above form, is used only exceptionally and does not constitute a material part of the food of the people, the malady is observed rarely or not at all. As regards the first point in this contention, there cannot be a single doubt entertained when we examine the pellagra-area; the second argument rests upon an extensive experience gathered from the various places within that area. Attention had already been drawn by Strambio to the fact that in the districts of Lombardy which formed the chief seats of the malady, most of the cases occurred in those villages where polenta of maize constituted the food of the inhabitants more or less exclusively. It was afterwards pointed out by Balardini that in Brescia and Bergamo, the head-quarters of the disease, polenta was consumed to so great an extent that the local production of maize did not suffice for the demand, large quantities of the corn having to be imported from adjoining provinces; while in those districts of the country where other cereals, more especially rice, took the place of maize, the amount of pellagra was very slight. It had been observed by Vallengaska¹ that, in some parts of the severely affected Venetian province of Belluno, where the potato had been introduced and had replaced maize as an article of diet, pellagra had diminished considerably. The statement was confirmed by Pertile, who added more generally that the disease was most severe in the lower parts of the province where the diet was almost exclusively polenta, whereas in the upper parts, where the inhabitants were better off and used other kinds of food as well, it was much less common. To the same effect we read in the official report of 1879:²

“La causa d’una relativa immunità nel Cadore e nell’Agordino viene spiegata del modo alquanto diverso d’alimentazione di quegli alpigiani ai quali la ricchezza dei boschi

¹ ‘Della Falcadina,’ Venez., 1842, p. 190.

² ‘La pellagra,’ p. 146.

rende meno disgraziata la vita, potendo essi, oltre della polenta, cibarsi di patate, d'orzo, fagioli e latticini in più larghe porzioni degli altri villici della provincia."

In the province of Novara (Piedmont), the chief seat of the disease is the district of Canavese, where polenta is the staple food; but in those parts of Piedmont (Biella, Varallo, Pallanza, Domodossola,¹) where the diet consists of polenta made from sound maize, and of chestnuts, wheaten bread, and potatoes as well, pellagra is either not seen at all, or it occurs in sporadic cases only. The same holds good for the country round Genoa and for the Ligurian coast. From the report of the Sardinian Pellagra Commission, which was far from being committed to the maize theory, we learn that 522 out of 626 pellagrous persons, or 83 per cent., lived on polenta alone, the rest having used other articles of food as well. Vignoli states that the frequency of the disease in the various affected districts of Tuscany is directly proportionate to the extent in which polenta has become the staple food; it had been previously shown by Chiarugi that the disease, on its first breaking out in Mugello and vicinity, had attacked only those villages where the people lived almost exclusively on maize, sparing those where wheaten flour, rye, or chestnuts entered into the diet. In like manner, for Roumania, Felix observes that there is little or no pellagra in those districts where the food is mainly other kinds of cereals and fish. Lastly I shall mention the fact dwelt upon by Chiappa, that pellagra is hardly known in great domains, chateaus, and the like occupied by rich proprietors, or in towns, where polenta is always more of a dainty than an article of diet.

(2) There is not a single fact to prove, or even to hint, that pellagra had been seen in any part of Europe before the introduction of maize; nowhere in that continent did pellagra show itself until maize became a staple food of the people. Evidence of this is furnished on comparing the date of the outbreak of pellagra with that of the introduction of maize cultivation into Europe. The plant, which is a native of the Western Hemisphere, is said to have been brought by Columbus to Spain, but it was not until after the first quarter of the sixteenth century that it was grown there to any

¹ *Ib.*, p. 47.

extent. The date of the introduction of maize into Italy falls about the middle of the sixteenth century; although it was not from Spain that it was brought but from Turkey (hence the colloquial Italian name "grano turco"), into which the plant had been introduced by the Spaniards shortly before. It is, however, from the middle of the seventeenth century that we have to date the general cultivation of maize in Italy, and it was not until the beginning of the present century that it began in France. It was introduced into Roumania and recommended to the people for cultivation by Niclaus Maurocordato in 1710.¹

(3) A change in the diet, the interdicting of maize altogether and the providing of other kinds of food instead of it, has in many cases effected a complete cure where the malady was in its early stage. It had been asserted by Casal that the first thing wanted to overcome pellagra was to change the food, and particularly to exclude maize from it; if adopted at the beginning of the disease this was the best means of getting rid of it. Cerri, at the instigation of the authorities in Milan, submitted ten well-marked cases of pellagra to a suitable diet of meat and bread (excluding polenta), and in the course of a year he thereby effected a perfect cure. Similar observations have been published by Brierre de Boismont, Roussel, and others.

§ 65. NOT DEPENDENT ON THE LOW NUTRITIVE VALUE OF A MAIZE DIET.

Against these arguments adduced in favour of the disease being caused by living on maize, a number of objections have been raised. More particularly it has been contended that the maize-zone, corresponding to those regions of the globe where maize is not merely cultivated, but also used as a material part of the people's food, extends very widely throughout the Eastern and Western Hemispheres, whereas pellagra is confined to only a few comparatively small districts of Europe; that even in these the disease did not appear until many years after the growing of maize had begun, the grain having been previously used by the people

¹ Theodori, p. 54.

as food without harm ; and, lastly, that sporadic cases of pellagra have been observed in various parts of France where maize is not a general article of food, and in particular had not entered into the habitual diet of the persons affected. Leaving the last of these arguments for future discussion, a certain weight attaches to the first two only in so far as they controvert the opinion held by many authorities,¹ that the essential cause of pellagra is *an exclusive or at least a preponderant diet of maize as such, by reason of its small nutritive value, and more particularly of the small amount of nitrogen that it contains.* This theory will be seen to be quite untenable when we consider : (1) that, although insufficient food may certainly induce chronic inanition and marasmus, yet the perfectly well-marked morbid state which is distinctive of pellagra is never developed under that influence ; (2) that those who fall ill of pellagra not unfrequently have the look of being well nourished at the beginning of the malady, the wasting which occurs in its subsequent course being the effect of organic disease and particularly of the intestinal affection ;² and (3) that large bodies of people who live solely on rice or potatoes remain quite free from pellagra although these articles of food are far behind maize in nutritive value, as the following analysis shows :

Table of Comparative Nutritive Values.

	Nitrogenous matters.	Carbo-hydrates.	Salts.	Water.
Wheat	186	655	17·2	140
Maize	100	744	16·9	139
Rice	78	781	3·0	137
Potatoes	17·9	215	9·7	755

If, then, there be some direct causal connexion between the use of maize as food and the production of pellagra, it must be a question of certain qualities of this nutritive

¹ Fanzago, Strambio, Verga, Paolini, Westler, Vignol, Morelli, Lusannae Frua, Leonardi, Bonfiglia, and others.

² See Sormani, p. 250.

substance which are not proper to its nature, but acquired by it under particular conditions, that is to say, a question of the grain having undergone a change through decomposition or disease. It had been conjectured by several of the earliest observers of pellagra, by Casal, Frapolli, Gherandini, and others, that the use of damaged maize (*mais guasto*) was at the root of the disease; and the attention of investigators was afterwards directed to the nature of this decomposition in the grain,—to the circumstances under which it took place, the kind of change which the corn underwent, and the connexion between grain thus altered and the pathogenesis. And if these inquiries have not yet led to a final solution of the question, yet the experimental evidence has shown with a high degree of probability, that *certain toxic substances, representing the proper cause of the malady, are developed in the course of the decomposition of Indian corn, and possibly under the influence of epiphytes on the corn.*

§ 66. DUE TO MAIZE CULTIVATED AND HARVESTED UNDER
UNFAVORABLE CIRCUMSTANCES.

Maize requires, for its full flourishing, a climate approximating to that of tropical or sub-tropical regions, and a strong loose soil, sand with loam being the best. The farther any locality is from these climatic and geological characters, the more imperfectly does the Indian corn ripen and the more readily does it undergo decomposition, the latter being particularly apt to occur when the grain is gathered and stored while it is still in a damp state. Roussel, Bataille, Michelacci, and others have pointed out that pellagra is quite unknown in those latitudes where the climate as a rule makes it possible for the crop to ripen to the full, as in the countries where maize is indigenous, and in others such as India and Nearer Asia; that the geographical distribution of this disease falls precisely within those latitudes where the crop is less thriving from want of the conditions above mentioned, the grain often failing to come to full maturity; but that even there, with certain precautions and careful management of the crop, it may be prevented

from spoiling, and that in this lies the explanation of the fact that many regions in temperate latitudes, where maize is grown and enters largely into the food of the people, are happily free from pellagra.

This origin of pellagra, from consumption of maize which had been cut before it was quite ripe, gathered before it was dry, stored in its damp state, and consequently become putrid, is the explanation that we meet with in Balardini, Facen, Assandri, Triberti, Tassani, Liberali, Girin, Pertile, Cambieri, the whole profession in Roumania, and the larger number of the more recent observers in general. It is remarked by Tassani that the districts in the province of Cremona which suffer most from pellagra are those in which the maize chiefly used is the so-called quarantin maize (*zea mays praecox*), or, in other words, the smallest variety of the plant. The late sowing, the lateness of the autumnal ripening and the consequent immature harvesting, the gathering of the corn in its wet state in damp weather, the bad storage (bad because this kind of maize is destined for the food of the poorer classes, and not for the market, like the large kind of autumnal or August maize),—all these things contribute in a marked degree to its rapid decomposition. Scheiber, who is himself an opponent of the maize theory of pellagra, calls attention to the fact that the Wallack peasantry of Transylvania who are free from pellagra notwithstanding their addiction to a diet of maize-polenta, had long ago learned from their Saxon neighbours a better way of tilling their fields and an improved husbandry of their crops of corn, so that they allow the maize to ripen as much as possible and then dry it on the ground and in barns; whereas the Wallack peasantry of Roumania, who are subject to pellagra, do not permit the corn to become ripe enough, and shoot it into pits where it becomes musty or spoilt.¹ Typaldos gives the following interesting information relating to the outbreak of pellagra in Corfu: The maize grown on the island usually thrives remarkably well, and supplies a good kind of sustenance; however, since about thirty years ago—the account dates from 1866, and the first appearance of pellagra in

¹ Theodori (l. c., p. 53) and Felix (l. c., p. 23) confirm this statement, particularly as regards the unripe state in which the maize is harvested in Roumania.

Corfu from 1839—vine-planting has extended very much in the island, almost displacing the cultivation of maize in some districts, so that large quantities of that grain have had to be imported from neighbouring countries to make good the deficiency, especially from Albania, Greece, Southern Italy, and the Danubian Provinces. The maize from the former group of countries is as good as that grown in Corfu, but the Roumanian maize is very often damaged and mouldy, partly in consequence of the long sea-transit; and Typaldos has satisfied himself that it is precisely this Roumanian product which constitutes the great bulk of the maize consumed in Corfu, and that a large part of the grain comes into market in a very damaged state.

§ 67. A BAD MAIZE HARVEST IS FOLLOWED BY AN INCREASE OF PELLAGRA.

Further support to this doctrine is given by the collected experience as to the influences above mentioned of *states of the weather and the soil* upon the goodness or badness of the maize crop and upon the amount of the sickness. In Italy it has been often observed, and not unfrequently at various points, that a remarkable increase occurs in the number of those attacked by pellagra—a sort of pellagra epidemic—whenever there has been a bad harvest in consequence of unpropitious weather, when the maize corn has been malformed, gathered half ripe owing to the peasantry being short of food, and stored or used in its wet state. Earlier authorities had already noticed these fluctuations in the amount of the disease from year to year,¹ and a series of observations has actually proved that those exacerbation-periods of pellagra had a remarkably close correspondence with the years noted for bad harvests and famine. Chiappa, Hildenbrand, Menis, De Rolandis, and others adduce in this connexion the experiences of 1755, 1801, 1815-17, 1822-23, 1829-30, and 1838. Tassani remarks that in 1830 those

¹ Among others Cerri ('Giornale,' l. c.), who observed a remarkable increase of the malady in Somma (province of Milan) in 1878, contrasting with its relatively small amount in 1876 and 1877.

villages in the Cremona district suffered from pellagra most in which the corn turned out to be of particularly bad quality, and that the same thing had been observed there in 1838 as well as at more recent periods. In Piedmont, as Girin and others inform us, pellagra is always most frequent in dear years, when the peasant does not wait for the full ripening of the maize, and an inferior kind of flour is made from the prematurely harvested crop; and the experience is the same from Ferrara in 1853-54, from Mantua¹ in 1873 (after heavy floods), and from the Modena² district in 1874, when the peasantry were obliged to live on bad polenta after the total failure of the crops the year before.

Besides being dependent on the weather, the thriving of the maize crop depends on the above-mentioned *conditions of soil*. We find accordingly that pellagra is particularly common in localities with a poor soil of sand, marsh, or clay, little adapted for growing maize, and upon which the grain is all the more likely to go wrong; of this we have information by Hammer from Lombardy, by Girin from the district of Canavese (Piedmont), and by Marchand from the department of Landes. Felix points out that in Roumania the people suffer most in the mountainous districts, where the maize ripens with greatest difficulty, and that the number of cases is in proportion to the goodness or badness of the yield of corn.

§. 68. THE PELLAGRA-POISON: EXPERIMENTAL EVIDENCE.

As the conviction steadily gains ground that it is not a diet of maize as such, but of *damaged maize*, that furnishes the cause of pellagra, the question necessarily forces itself upon us, What is the decomposition-change in the maize with which the pathogenic effect is bound up, or what is it in spoiled maize that constitutes the proper *pellagra-poison*? The conjecture had already been thrown out by Sette that it is an affair of a toxic parasitic mould, as in ergotism; and that idea has been taken up and followed out by Balardini. On musty maize the latter found a greenish-coloured mould,

¹ See Parola, l. c., p. 753.

² Martinelli, Maragliano.

to which he gave the name of "*verderame*;" it was recognised by Cesati as belonging to the genus *Sporisorium* and named *Sporisorium maidis*. Toxic experiments which Balardini made upon men and animals with maize so affected gave results which seemed to justify that conjecture as to the origin of pellagra. Fowls which were fed on such maize became thin, lost their feathers, became affected in their power of movement, and died with still other nervous symptoms. In the human subject there occurred burning in the throat, digestive troubles, and diarrhœa. Although Rezzi, as reporter of the commission appointed by the Istituto Lombardo to test the discovery of Balardini, gave an unfavorable verdict, chiefly on the ground that the *verderame* was very often found on maize in many other regions, such as Southern Italy, which were quite free from pellagra; yet Balardini's theory was adopted by Roussel and Costallat, while Tardieu¹ gave a favorable opinion on it in name of the Commission of Inquiry nominated by the French Minister of Agriculture.

The question seemed thus to be settled, when Lombroso² made it the subject of exact investigation anew. He confirmed the result of Balardini's toxicological experiments with decomposed maize, and indeed, in his experiments on the human subject, the characteristic phenomena of pellagra in the skin and nervous system came out more decidedly than in those made by Balardini. In fowls he observed diarrhœa, casting of their feathers, and death; in rats, wasting, choreiform movements, contractures, and a fatal issue as in fowls; in healthy men, after a prolonged course of a tincture prepared from *mais guasto*, loss of appetite, vomiting, diarrhœa, desquamation of the cuticle, giddiness, dilatation of the pupil and mal-nutrition.

But at the same time he proved that the view of Balardini, according to which the *verderame* was the proper toxic principle, must have been based on an error, inasmuch as that fungus is generally speaking very rare; so rare, indeed, that he did not succeed in finding it upon maize in the course of a journey through the whole of Lombardy, while

¹ His report is reprinted in Costallat's paper.

² In papers (II) and (III) under his name in the list at the end of the chapter.

two of the foremost botanists in Italy who made a search for it, discovered it on only a few grains of corn now and then. It is probable, as Lombroso conjectures, that Balardini had confounded the *Sporisorium* with *Penicillium glaucum*, which is certainly very common on musty maize, although Lombroso's experiments with it prove it to be perfectly harmless.

In view of these facts, both positive and negative, Lombroso considered himself justified in concluding that the toxic principle was not present in maize-corn in the form of a parasitic mould, but that certain substances were developed in the parenchyma of the decomposing corn, which had a specifically toxic action as above, and were the proper cause of pellagra. Further inquiries made by him, in conjunction with Dupré, Brugnattelli, and Erba,¹ on these decomposition-products formed in maize, have shown that the chief thing is the occurrence of a fatty oil (maize-oil), and an extractive substance (named by Lombroso "pellagrozein" and by Erba "maizina") which are never found in sound maize; that the decomposition-products can be artificially produced in the corn when it is exposed to fermentation; that these matters are much more powerfully toxic when produced in the hot season than when obtained in cold weather; and that their effect upon the animal body is to induce a series of phenomena which do not indeed bear the perfect impress of the pellagrous group of symptoms, but are yet a decided expression of some severe cerebro-spinal affection, and reveal many analogies with the phenomena observed in pellagra.

In forming a judgment on these admirable studies of Lombroso, there are two things to be kept in mind: firstly that the toxicological experiments were made on the lower animals, which react to poisons in a different way from man, and differently, too, in their own several classes; and secondly, that they deal with acute poisoning, whereas pellagra is a disease depending upon a chronic toxic effect.

There have naturally not been wanting objectors to the discoveries of Lombroso and to his theory of the genesis of pellagra—among them Gemma, Bellini, Lussana (III and IV),

¹ These inquiries will be found in Lombroso's papers (IV), (V), (VI), and (VII).

Ciotto, and Bonfigli;¹ Biffi also, the reporter of a Commission of Inquiry appointed by the Instituto Lombardo, has spoken very doubtfully of them. On the other hand, the experimental results obtained by Tizzoni and the Roumanian physicians (Felix) entirely agree with those of Lombroso. But more especially Cortez, working at the instigation of Husemann and in association with him, has carried out a considerable number of very exactly planned experiments with the toxic substances obtained from maize by Lombroso and Erba, which confirm Lombroso's conclusions in almost every point; so that there cannot be the slightest doubt as to the trustworthiness of the latter. At the same time it must remain an open question how far the formation of these toxic substances in decomposed or fermenting maize is dependent on the presence of the lower organisms which are always found to coexist.

§ 69. AFFINITIES TO ERGOTISM.

If, then, the question of the cause of pellagra has been settled by the labours of Lombroso, not perhaps altogether finally, but, in my opinion, very nearly settled, the theory expounded in the foregoing sections will receive additional and very material support from the *similarity which pellagra shows to other analogous toxic diseases and particularly to ergotism*. Strambio, Jansen, Hildenbrand, and other of the older observers, as well as Rayer among the recent, have called attention to that resemblance. Of special weight in this regard is the declaration of Hebra:² "I have seen a large number of cases of pellagra. The type of the disease has unquestionably the closest resemblance to other toxic conditions induced by decomposed vegetable matters; only the effect is slower (but not the less profound on that account), and the course more protracted. One is involuntarily reminded of ergotism."

¹ In an article ('Gaz. med. Lombard,' 1880, No. 47) directed against Lussana and Ciotto, Lombroso points out the want of judgment with which their toxicological experiments had been devised.

² In the 'Handb. der spec. Pathol.' (edited by Virchow), III, i, 205, note 2.

§ 70. OBJECTIONS TO THE MAIZE-THEORY ANSWERED.

Against this theory of the origin of pellagra from the consumption of decomposed maize, there have been two objections raised which call for a brief discussion. The first of these is, that maize is grown and used as a popular article of food in many localities which are not better adapted for its cultivation than the pellagrous districts themselves, and yet the disease does not occur in them. The second objection is that in many parts of France, as we have seen (p. 234), sporadic cases of pellagra have been observed, which cannot be brought even remotely into connexion with poisoning by maize.

As regards the first point, special stress has been laid on the fact that maize is largely cultivated and used as food in Burgundy, in the Bresse, and in Franche-Comté, while pellagra is quite unknown in these provinces. It has been shown, however, by Roussel and Costallat that the newly gathered maize in all these localities is thoroughly dried, or rather scorched, before it is stored or put to use, and that any decomposition of the corn is thereby prevented. The same practice is observed, as I have had frequent opportunities of satisfying myself, also in Southern Italy, where moreover, polenta of maize figures to a much smaller extent than in Northern Italy. Salas, in his interesting work on pellagra, informs us that in Mexico, as well as in America generally, the maize is shelled out directly after harvesting, and the corn is then thoroughly dried in the sun. No doubt damaged maize sometimes occurs even there, especially towards the end of winter; but it is usually made into food for the horses, or mixed in small quantities with sound maize by the poorer class of people, and in that case always subjected before being cooked to certain processes which render it harmless.

Pseudo-pellagra.—The second objection is substantially met by the argument that most of the cases of so-called “sporadic pellagra” clearly rest on errors of diagnosis. Roussel has called them “unités factices,” or arbitrarily made-up groups of symptoms in which nervous and psychical

affections are included with disorders of the digestive organs and with morbid appearances in the skin, and the name of "pellagra" given to them. Billod went so far as to assert that it is a mental disorder that we have to do with mainly in pellagra, and that when affections of the digestive organs and of the skin are added thereto, we have "pellagra" complete; others such as Hardy,¹ have confounded pellagra with alcoholism; so that Tardieu was perfectly justified in declaring that there never had been such confusion of ideas among French physicians as on the subject of pellagra. Moreover, these authorities must admit, as regards the cases of "sporadic pellagra," that there was never more than a resemblance between them and the endemic disease; and Roussel, accordingly, found it necessary to speak of them as "pseudo-pellagra." This is the judicial standpoint assumed by nearly all the more recent observers, such as Chaussit, Vernois, Pellizari, Brierre de Boismont (II), and various others; Dejeanne, who has made a very full collection of all the observations relating to this matter, and subjected them to a thorough scrutiny, says that "these are maladies differing widely among themselves, and all of them very different from endemic pellagra not only in the etiology, but also in the nature and concatenation of the symptoms."

It should not be denied, however, that sporadic cases occur by no means exceptionally outside the foci of pellagra, just as undoubted sporadic cases of leprosy have been seen outside the leprosy centres. The question had already been put by Bouchut² in this connexion, whether other kinds of grain as well, such as oats, may not under certain circumstances suffer changes like those of maize, and thereby give rise to pellagra in localities where maize is not grown or not used as food. To the like effect Husemann says:³ "There is absolutely no reason why a similar toxic principle should not develop in any other substance, which approximates to the grains of *Zea Maïs* in chemical composition, although it may not be actually derived from that plant. It is no remote possibility that a process of decomposition may be set up in the flour of other kinds of grain under the influence of defi-

¹ 'Traité de pellagre,' Paris, 1866.

² 'Arch. gen. de méd.,' 1867, Nov., p. 503.

³ L. c., p. 272.

nite external conditions, leading to the formation of peculiar matters whose gradual introduction into the organism would produce phenomena either identical with those of pellagra or presenting at least close analogies thereto." It is remarkable, moreover, that sporadic cases of pellagra have never occurred, or at least never been observed hitherto out of France.

§ 71. NOT CONTAGIOUS ; DOUBTFULLY HEREDITARY.

The view held by some of the first observers of pellagra, that the disease spreads by way of *contagion*, is opposed in the most decided manner by all the later investigators both on positive and on negative grounds. The almost absolute immunity which the urban population enjoy from pellagra, notwithstanding their active intercourse with the inhabitants of the surrounding country, should be sufficient of itself to decide the question in a negative sense.¹ While there is unanimity as to non-contagiousness, there is also a positive agreement among the greater number² of observers that pellagra is transmitted by heredity, although the views diverge on the point whether we have to do with inheriting the disease itself, that is to say, with a congenital dyscrasia—"si nasce pella-grosi," says the Piedmontese Commission—or with an inherited specific predisposition, such as scrofula is, or finally with a state of congenital feebleness referable to the deteriorated health of the parents, and serving to increase the susceptibility of the individual to the specific influences by lowering his general power of withstanding influences from without. The occurrence of the disease among infants at the breast might have been taken as favouring the first mentioned kind of conveyance, were it not that we learn from several authorities, such as Lussana and Frua, that even sucklings are fed with polenta.

The great discrepancies among the data to hand do not permit us to decide how much significance for the spread of the disease attaches to this factor. The Piedmontese Commission found only 189 cases out of 927, or 20 per cent., clearly hereditary; according to Lombroso, heredity could be proved for only 74 in 472 cases or about 16 per cent.; Mara-

¹ See 'La pellagra,' pp. 344—51.

² Felix (l. c., p. 17) denies the hereditary transmission of the disease.

gliano found heredity pronounced in 26 out of 150 cases observed by himself, or 22 per cent. On the other hand, an examination of 815 pellagrous inmates of lunatic asylums showed that 415 of them, or nearly 50 per cent., came of pellagrous parents. Boudin has ascertained that in 657 married couples, with 740 pellagrous children, both husband and wife were pellagrous in 15 per cent., the husband only in 24 per cent., the wife only in 27 per cent., while in 18 per cent. both parents were healthy, and had several pellagrous children, and in 16 per cent. the parents were healthy with only one child pellagrous.

This question naturally cannot be decided for certain unless we were to ascertain how many of the pellagra patients who had been born of pellagrous parents, were withdrawn from the pathogenic influences at birth, and developed the disease although kept remote from them; but I do not find any figures of that kind in the reports to hand.

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Acrodynia (mal des pieds et des mains, erythème épidémique).

§ 72. CLINICAL CHARACTERS. HISTORICAL OUTBREAKS.

Under these and other names the French physicians have described a disease which appeared in epidemic form in 1828 and 1829 at several places in France, being very widely diffused in Paris. It shows so many striking analogies with ergotism, and even more with pellagra, that it may find a place in the meantime along with these toxic diseases, although there is still much that is doubtful as to its essential nature and its causes.¹

Symptoms.—The onset of the disease is usually marked by signs of more or less intense irritation of the gastric and intestinal mucous membrane, a feeling of weight in the stomach, sickness and vomiting, and by loose or even dysenteric stools which continue in many cases until near the end of the attack and serve to reduce the patient greatly. With these symptoms, there are usually associated redness and puffiness of the conjunctiva, and an œdematous swelling of the face, which is for the most part very transitory. After a few days (five to twelve) formication comes on, and flying pains in the hands and feet like the pricking of needles, often very acute, or intense burning in the soles of the feet; hyperæsthesia exists over large areas of the skin, which increases with the external heat and makes even the slightest pressure unbearable. After these phenomena of morbidly increased sensibility have passed off, or in some cases, from the very first, the patients suffer from anæsthesia, especially in the soles of the feet, like the anæsthesia of tabes dorsalis. Associated with the development of these nervous symptoms there is usually an outbreak of an erythematous or erysipelas-like exanthem, which spreads mostly over the hands and feet, but not unfrequently over a large part of the extremities and even over some parts of the trunk; the skin at these spots becomes gradually thickened and wrinkled.

¹ Bayle, 'Revue méd.,' 1828, iv, 445; Chardon, *ib.*, 1830, iii, 51, 374; Cayol, *ib.*, ii, 48; Chomel, *ib.*, iii, 485; Genest, 'Arch. gén. de méd.,' 1828, xviii, 232, 1829, xix, 63, 357; Longueville, *ib.*, 1828, xviii, 310; Villeneuve, *ib.*, 122, 311; Hervez, 'Journ. gén. de méd.,' 1828, cv, 15; François, *ib.*, 360; Montault, *ib.*, cvi, 170; Prus, *ib.*, 385; Kuhn, 'Bullet. des. sc. méd.,' 1828, xv, 252; Sédillot, 'Gaz. méd. de Paris,' 1833, 266; Andral, 'Gaz. des hôpit.,' 1833, Septbr.; Clairat, 'Considér. sur la malad. épidémique, &c.,' Par., 1829; Miramond, 'Diss. sur l'affection épidém., &c.,' Par., 1829; Rue, 'Essai sur la maladie qui a régné épidémiquement, &c.,' Par., 1829; Ratier, 'Clinique des hôpit.,' 1828, 20, Novbr.

and at length discoloured to a dark brown or black in consequence of an abundant deposit of pigment, the discoloration being greatest in the region of the nipples, over the lower part of the abdomen, on the neck and in the folds of the axilla, elbow, groin and ham. In cases of a severe type paroxysmal seizures of cramp, or evidences of paresis of the extremities, will occur in the subsequent progress; the limbs are kept continually bent in a state of tonic spasm, or there is inability to grasp and hold objects or to walk straight. If these nervous attacks should have persisted for some time, the affected limbs waste and there occurs œdema of the thighs and legs, and sometimes even general anasarca. The disease runs its course without fever; it lasts from a few weeks to several months, relapses contributing materially to its long duration. It was only rarely, in the case of old and enfeebled persons, that it ended fatally, and then mostly in consequence of long-continued diarrhœa. Recovery was always slow and there was always weakness present for some time after, and a feeling of stiffness in the limbs that had been affected. Post-mortem examination did not throw the faintest light on the nature and seat of the disease; the most careful naked-eye examination of the spinal cord and nervous system in general was absolutely fruitless; "l'acrodynie," said Andral, "est une de ces maladies que l'anatomie pathologique ne peut éclairer en aucune manière."

History.—Acrodynia showed itself first in the winter of 1827-28 at Paris, where it was scattered over the whole city in isolated cases, but was most abundant in the Quartier Hotel de Ville and in the Rue Petits Augustins. Towards spring the number of cases increased,—so considerably, indeed, that they numbered 40,000 by the end of summer. Meanwhile news had come of its prevalence at Meaux, Troyes, St. Germain-en-Laye, Noyon, and other towns in France. During the autumn and winter following the cases again became sporadic, but in the spring of 1829 the disease resumed its epidemic character in Paris, and broke out simultaneously at Coulommiers, Soisy-sous-Etiolles, Montmirail, and other places, continuing to be prevalent until the approach of autumn and dying out entirely in the winter.

All observers were agreed that acrodynia was a disease quite unknown in France previous to its appearance there in 1828; and, in so far as we have any published epidemiographical information from former centuries or from the first part of the present, or in so far as such is known to me, I must not only confirm that opinion, but, in agreement with Andral who spoke of acrodynia as "une maladie inconnue jusque là dans le monde," I must say further that there are absolutely

no prior observations on the disease, be they descriptions or mere indications, in the literature of medicine generally.¹ Subsequent to 1830, accounts of sporadic cases of acrodynia were published from various parts of France,² which show, however, very material deviations from the type of the disease as deduced from the epidemic of 1828-30. It is only the isolated cases seen by Barudel³ in 1859 among the troops in Lyons that bear the perfect impress of acrodynia, and perhaps also the case observed by Roucher⁴ at Setif (Algiers) in a French soldier, who had, in addition to other characteristic symptoms, the burning very well marked in the soles of the feet and afterwards in the thighs. We may also include with these the small epidemic which occurred in 1874 in a regiment at the camp of Satory near Versailles, described by Bodros⁵ (who had fourteen cases under his own notice in two months), although the gastric symptoms were less marked, and the hyperæsthesia, cramps, and dropsical phenomena entirely wanting.

Of recent years epidemics of acrodynia have been observed also in other parts of the world—in several *Belgian* prisons in 1846, among the French troops at the seat of war in the East in 1854 (*Crimea* and *Constantinople*), and among Mexican and French (Algerian) soldiers in *Mexico* in 1866.

In Belgium the epidemic of the disease occurred in December, 1845, but it had been preceded, according to Vleminckx,⁶ by sporadic cases during 1844 and 1845 in the

¹ The account by Santo Nicoletti of a disease observed in a number of soldiers at Padua in 1806, the same disease, according to this authority, from which S. Marino and Savigliano had suffered in 1762, and had been described by Ozanam ('Hist. méd. des malad. épidém.,' 1835, iv, 242) under the name of "Pédionalgie," does not in any way point to acrodynia (see also Corradi, 'Annal. delle epidemie occorse in Italia,' iv, 582). The same remark applies to McGregor's account of a disease of the feet observed among the English troops in Spain in 1812 and 1813 ('Med.-Chir. Transact.,' 1815, vi, 381).

² Raimbert ('Revue méd.-chir.,' 1848, Mai); Chéveriat ('Gaz. des hôpité,' 1850, 71), and Beau (ib., 1862, 302), for Paris; Saucerotte (ib., 716) for Lunelville; Ganiez (ib., 1878, 226), for Darney (Vosges).

³ 'Mém. de méd. milit.,' 1861, i, 367. ⁴ 'Union méd.,' 1866, Novbr., 409.

⁵ 'Mém. de méd. milit.,' 1875, Septbr. and Octbr., 428.

⁶ 'Bull. de l'Acad. de méd. de Belgique,' 1846, v, 410, on the information of Tosquinet and Stanquez for Brussels, Chamberlain for Namur, and Mareska for Ghent. See also the account by de Maeyer in the 'Annal. de la soc. de méd. d'Anvers,' 1846, p. 443.

St. Bernard House of Correction at Brussels, which institution, along with the prisons of Ghent and Namur, formed the chief seat of the epidemic. The type of disease, except as regards the disorders of digestion which were the exception in this epidemic also, corresponded perfectly to the description given by the French physicians; in one case gangrene of the lower extremities was observed, and in another gangrene of the scrotum. In the prison at Brussels the disease assumed a particularly severe form, thirty-three dying out of the 288 persons attacked by it; it may be inferred, however, from the narrative, that it was complicated with typhoid in many cases. In the cases observed in the Army of the East, estimated by Tholozan¹ who reported upon them at 500 to 600, the symptoms of intestinal disorder were less marked than in the first French epidemic; rarer also were the conjunctivitis and the affection of the skin, while œdema, which always preceded the nervous symptoms, was uniformly present. The epidemic in Mexico, according to Laveran,² was confined exclusively to a division of Mexican *franc-tireurs* and to a battalion of Algerian *tirailleurs*, the disease running its course with exactly the same symptoms as at Paris in 1829.

§ 73. AFFINITIES OF ACRODYNIA WITH ERGOTISM AND PELLAGRA: THE FOOD SUSPECTED.

The French practitioners who witnessed the epidemic of 1828-29 were almost unanimous in recognising *a similarity between the phenomena of acrodynia and those of ergotism and pellagra*; and some of them, such as Ratier and Kuhn, expressed their conviction that the disease was a toxic process, just as in those two diseases. Neither did the Belgian physicians overlook that correspondence in the symptomatology, Stanquez declaring that the malady vividly recalled raphania. It would appear that subsequent observers of acrodynia had been impressed with the same view, for they gave special attention, in their search after the cause, to the

¹ 'Gaz. méd. de Paris,' 1861, p. 647, *seq.*

² 'Mem. de méd. milit.,' 1876, p. 119.

quality of the *food* served out to those who fell ill, particularly the flour and bread. Although the inquiries directed to that point led only to negative results both in the first and in all subsequent epidemics; yet the possibility is not excluded of acrodynia being a poisoning by the food similar to that which occurs in the toxic diseases already mentioned. Stanquez, after assuring us that the most careful examination of the flour used in the House of Correction at Brussels did not reveal the slightest trace of decomposition in it, goes on to remark: "Ce qui est incontestable, c'est qu'il y a intoxication."¹

It is a noteworthy fact, moreover, that the disease in the epidemic of 1828-29 was prevalent almost exclusively among the poorer classes, particularly in *garrisons, hospices, prisons*, and such like confined places, the inmates of which were subject to a uniform diet, and in which the disease no sooner appeared in one case than it spread to the whole, or nearly the whole of them. Thus in the Caserne de l'Oursine 300 men of a battalion were seized with acrodynia at once; in the Caserne de Faubourg du Temple not one soldier escaped it; in the Marie Thérèse Hospice thirty-six out of the forty inmates fell ill. In Belgium the epidemic was strictly limited to prisons, and in Brussels the interesting observation was made that all those convicts escaped who were at work outside the House of Correction, that is to say, in the open air. In the Crimean War and in Mexico, the malady occurred among certain divisions of the troops only, a circumstance which would seem to make quite untenable Laveran's notion that it was the hardships of the service which properly caused the disease. There is, accordingly, some obscurity still hanging over the cause of acrodynia, and I shall not venture to say whether it may not be dispelled in the end by the discovery of something toxically injurious in the food.

¹ The attempt made by some authorities to identify acrodynia with dengue is quite mistaken. Also the opinion expressed by Le Roy de Méricourt ('Bull. de l'Acad. de méd.,' 1865-6, xxxi, 53) that the Paris epidemic of 1828-9 was an affair of *trichinosis* I take to be groundless; not merely the whole train of symptoms, but the mode of spreading in acrodynia, tells decidedly against that view.

Pelade or the Colombian Maize-Disease.

§ 74. TOXIC EFFECTS OF ERGOT OF MAIZE ON MEN AND ANIMALS.

According to Roulin,¹ there occurs in Colombia, particularly in the provinces of Neyva and Mariquita, a disease of maize caused by the ergot mould—*mais peladero*—by reason of which the corn acquires properties injurious to health. In man, as a consequence of using the diseased grain, the hair comes out—an occurrence all the more remarkable that baldness in those parts is rare even in old men—and sometimes the teeth get loose and fall out; but there are never the nervous or gangrenous incidents peculiar to ergotism. Animals, such as pigs and mules, suffer in the same way from eating ergotised maize; but in these there have been noticed also paresis and wasting of the hind legs. Fowls fed on *mais peladero* lay eggs without shells, the explanation of this, in Roulin's opinion, being that the egg is expelled by the tetanic contraction of the oviduct before the calcareous envelope has had time to get deposited round it. Pelade, he says, never occurs in Peru or in Mexico; and that statement is confirmed, as regards the latter country, by Celle.² There have been no accounts of this disease from Colombia³ more recently.

In connexion with the effects of the ergot-parasite of maize, Heselbach's⁴ observation is worth noticing, that eleven cows cast their calves after eating diseased maize—he speaks of *Ustilago maidis*, but it was probably ergot of maize—and that abortion occurred in two pregnant bitches to which he gave the diseased corn pulverised. Estachy⁵ also has recently published observations on the power of ergot of maize to bring on labour.

¹ 'Journ. de chimie méd.,' 1829, v, 608; 'Transact. méd.,' 1830, i, 420.

² 'Hygiène prat. des pays chauds,' Paris, 1848, p. 173.

³ Husemann having had his attention called to Roulin's paper in the course of his inquiries on the effects of decomposed maize ('Arch. für experiment. Pathol.,' 1878, ix, 276), sent to Santa Fé de Bogota for information about pelade, but he has learned nothing hitherto.

⁴ 'Magazin der Thierheilkde.,' 1860, p. 211.

⁵ 'Bull. gén. de thérap.,' 1877, xciii, 85.

CHAPTER VII.

MILK SICKNESS AND THE TREMBLES (SICK STOMACH, SWAMP SICKNESS, OR MILK DISEASE).

§ 75. "THE TREMBLES," AN ENZOOTIC DISEASE OF CATTLE IN THE UNITED STATES.

In some parts of the United States a disease occurs among herbivorous mammals, mostly among cattle and less frequently among sheep, horses and deer, to which the colloquial name of "the trembles" has been given in consequence of the characteristic symptom of a kind of paralytic weakness and spasm-like trembling in the movements of the body.¹

Symptoms of "the trembles."—It has been noticed in the more slightly affected animals that, besides refusing their food they had difficulty in moving, and were easily made to tremble, the trembling being considerably aggravated when the animal's movements became more vigorous. At a more advanced stage of the malady, the weakness reached such a degree that the animal could not keep on its feet, the

¹ I subjoin an alphabetical list of all the papers on this subject known to me:—Carson, *ib.*, 1880, Octbr., 299; Coleman, 'Philad. Journ. of Med. and Phys. Sc.,' 1822, Aug., 322; Crookshank, *ib.*, 1826, Aug., 252; Crooks, 'Philad. Med. and Surg. Reporter,' 1873, July 22; Drake, 'Notices concerning Cincinnati,' *Cinc.*, 1810; Forry, 'The Climate of the U.S. and its Endemic Influences, &c.,' New York, 1842; Graff, 'Amer. Journ. of Med. Sc.,' 1841, April, 351 (the leading authority); Haines, 'Philad. Journ. of Med. and Phys. Sc.,' 1822, Aug., 331; Haller, in 'Transact. of the Illinois State Med. Soc.,' 1856; Lea, 'Philad. Journ. of Med. and Phys. Sc.,' 1821, May, 50; Lewis, 'Transylvania Journ. of Med.,' 1829, May, 241; McCall, 'Amer. Med. Recorder,' 1823, vi, 254; Minturn, 'Med. Times and Gaz.,' 1857, April, 420; Ref. (I), in 'Transylv. Journ. of Med.,' 1829, Febr., 145; Ref. (II), in 'Transact. of the Kentucky State Med. Soc.,' 1868; Ref. (III), in 'Philad. Med. and Surg. Reporter,' 1870, July, 102; Seaton, 'Philad. Med. Examiner,' 1842, Nr. 10; Shelton, 'Transylvania Journ. of Med.,' 1836, April; Simpson, 'On Milk Sickness,' Lexington, 1839; Smith, 'Boston Med. and Surg. Journ.,' 1868, Jan., 471; Sutton, in 'Transact. of the Amer. Med. Assoc.,' 1858, xi; Wright, 'Amer. Med. Recorder,' 1828, Apr., 401; Yandell, 'Transylvania Journ. of Med.,' 1828, Aug., 309.

whole body trembling violently, and the head tossing from side to side continually; sometimes the beast would be thrown down in a heap by well-marked convulsions, never to rise again. The muscles are kept rigid, those of the belly being contracted to the utmost; not unfrequently there is vomiting, the animal emits a peculiarly fetid breath, the eyes are dull and blood-shot, and death occurs, in most cases after a few hours of illness, with symptoms of extreme dyspnea. If an animal severely affected attempts any violent exertion, such as running fast, it collapses suddenly and expires in a few minutes. On examining the carcasses of animals that had been killed,¹ Graff found the cerebral sinuses very much distended, the veins of the brain-membranes choked with dark fluid blood, the pia mater clouded and covered with a more or less copious purulent exudation, and the brain remarkably soft. There were also traces of inflammation in the spinal membranes, the cord itself being very vascular. The stomach and intestines were extremely contracted, the mucous membrane of the stomach and small gut injected; lungs, liver, spleen and kidneys full of blood, the spleen sometimes swollen to twice its volume, and, like the liver, remarkably soft; the blood dark and perfectly fluid, without a trace of clot anywhere.

Geographical area.—This disease is mostly found in the Prairie States of North America (*Indiana, Illinois, Ohio and Missouri*), and in the more western of the Central States (*Tennessee, Kentucky and Virginia*). Sporadically, it has been seen also in Alabama, Georgia, and South Carolina; but in North Carolina, where it used to be prevalent especially along the course of the Yadkin, it has now completely disappeared before the thorough tillage of the soil.

§ 76. HISTORICAL NOTICES OF THE TREMBLES.

The earliest *information of the occurrence of the trembles* dates from the beginning of last century. The French missionary bishop Hennepin, who at that time penetrated along the rivers far into the western parts of North America, makes mention of the disease; but, the first precise accounts of it come down to us from the time when European colonization began to reach the Western States, general attention having

¹ The examination related to carnivorous animals which Graff had poisoned with the flesh of cows dead of the trembles. According to the brief notes given by him and McCall similar changes would appear to have been found in the cattle primarily diseased.

been directed to the disease by the frightful devastation that it made among the herds of the first settlers. To escape from this plague among their cattle, whole communities broke up before they had well acquired a firm footing in a locality; many fruitful tracts of country stood long unoccupied on account of it; and although the malady has become considerably less common in more recent times, it is still prevalent in a destructive form among the herds of cattle in many regions, such as Monroe County, Eastern Tennessee, (according to Carson). Not unfrequently, it breaks out as an epizootic, extending to many herds in some years and in other years only to a few.

§ 77. NATURE OF THE LOCALITIES WHERE THE TREMBLES
BREAK OUT.

Although this disease of cattle is found at all *seasons* and under every kind of *weather*, it is particularly common in summer and autumn, more widely spread in hot and dry weather than in damp, and most prevalent, accordingly, in years of drought.¹ Again, it has an unmistakable connexion with certain *localities*, or with *pasturages* of a certain kind. The experience of all affected districts goes to show that the cattle do not take the disease unless when they graze on particular ground, usually within a small area, closely circumscribed and situated in a deep valley or in the hollow of a meadow. They remain well so long as they are kept away from such spots; the fencing of these places, accordingly, gives perfect protection to the herd, and if the animals break through the fence, the consequence is that new cases of the disease occur among them. At many places it has been remarked that the disease has occurred among the cattle of some one farm only, all the other animals in the neighbourhood keeping perfectly free from it. Forry observes that nowhere and at no time has the focus of disease been known to enlarge its boundaries beyond the originally infected spot, that these centres of the enzootic have often undergone contraction but never expansion, that the malady has never

¹ Crookshank, Lea, McCall, Coleman, Minturn, Yandell, Haller, Thompson (in Haller), Simpson, Shelton, &c.

broken out in new districts, but only in those where it had existed from the first. Usually, as we have said, these foci of the disease are very limited in area ; it is only exceptionally that they extend over large tracts of country, as on prairies like those of Indiana, where the enzootic occurs for nearly a hundred miles continuously on a narrow strip along the banks of the Wabash.

§ 78. POOR OR FALLOW LAND FAVORABLE TO THE TREMBLES.

It is impossible to decide, so far as inquiries have gone hitherto, whether certain *peculiarities of soil* are to any extent accountable for this clinging of the disease to particular points. The dampness or dryness of the ground has no significance in this matter ;¹ it would appear to be of greater importance that the land should be *arable* or *cultivated*. Graff observes that there is something peculiar in the character of the enzootic districts in Edgar County, Indiana ; that they lie rather higher than other ground in the neighbourhood, being situate on ridges ; that their soil is in general poor, and their growth of wood sparse and stunted, such localities presenting now and then the appearance of a heath ; on the other hand, the disease never occurs in localities where there is a luxuriant growth of grass. All observers² agree that animals catch the complaint only in ranges of woodland or on land that has not been tilled, that regular tillage affords a sure protection against the malady, the cattle on well-tended meadows never taking it, and that allowing the soil to lapse into an uncultivated state is not unfrequently followed by reappearance of the old noxious property in the pasture.

§ 79. THE TREMBLES PROBABLY DUE TO A TOXIC SUBSTANCE IN THE PASTURE.

There can be no question that in this disease of cattle we have to do with a *toxic effect*, and that the poison must

¹ Coleman, Simpson, Yandell.

² Dixon (quoted by Lea), Coleman, Lewis, Simpson.

be in the soil, or rather *the produce of the soil*. But no certain proof has been given as yet of the nature of this poison. The conjecture thrown out by some observers that it is *poisoning by miasma* which underlies the disease, is quite untenable; at least we should have to regard it as something new in the history of miasmata if they can be kept within bounds by a fence. There is equally little likelihood in the notion¹ that some *mineral poison*, such as cobalt, may exist in the springs at which the cattle drink, having soaked out from the soil, and that this is the true cause of the malady. Against that assumption there is the fact that the symptoms are not in the least like those of arsenical poisoning; and we have further to bear in mind that the water from the presumably poisoned sources has often been drunk by men, especially by the cattle tenders, without any injurious effects ever following; while lastly there is a complete refutation of it in the results of Graff's exact inquiries, according to which no metallic substances are to be found in the suspected springs beyond a small amount of iron and a trace of copper, and certainly no trace of arsenic or salts of arsenic, which metal he had also sought in vain for in the soil of the diseased spots.² The most likely and most rational theory is that the disease is due to some *vegetable poisoning*; at all events, there is nothing opposed to this in anything that we know of its distribution, its way of breaking out and disappearing again under the circumstances above mentioned, or of its symptoms. But the decisive proof of this—the detection of the plant which contains the poisonous principle—is not yet forthcoming, despite the most careful search.

Some observers³ are inclined to the opinion that it is an affair of poisoning by one of the numerous toxic umbelliferæ which grow in such enormous quantities in the uncultivated parts of the Western States; others⁴ believe that the malady is a consequence of eating a species of *Rhus* (*Rhus toxicodendron*, the poison-oak). An opinion has lately

¹ Adopted by Shelton, Crookshank, Seaton and Haller.

² All the more recent observers pronounce against this notion of the trembles being caused by metallic poisoning.

³ Ref. I in the list above given.

⁴ Drake, Owen (quoted by Sutton), and others.

been expressed¹ that the poisoning is caused by a fungus belonging to the class of Coniomycetes, which the animals consume with their fodder.

§ 80. "MILK-SICKNESS" IN MAN CORRESPONDING TO THE TREMBLES IN CATTLE.

In all those regions where the cattle-disease known as "the trembles" is indigenous, a disease occurs in the human subject which perfectly resembles that malady in its symptoms, or, in other words, it has the same character of being a toxic state induced by a powerful vegetable poison.

Symptoms of the "milk-sickness."—The disease begins usually with pains in the head and limbs, a general feeling of weakness, disorders of digestion, and a peculiar fœtor of the breath; along with these symptoms there are anxiety, restlessness, great irritability, not unfrequently some slight confusion of ideas and an eager condition, while the movements are unsteady and tremulous, and the skin cold and wrinkled. Then, after a shivering fit and a hot fit following it, the patient vomits a soapy or greenish-yellow substance, not unfrequently bloody mucus, and, in the worst cases, blood presenting the appearance of coffee-grounds; the vomiting is attended by a feeling of oppression or burning pain in the region of the stomach, and of intense thirst and a desire to slake it by cold water. At the same time the bowels are constipated, in most cases completely, and the constipation is difficult to move. The pains in the neck and limbs increase, the tongue becomes dry and red, and often swollen to such an extent that it completely fills the mouth, being marked by the impressions of the teeth and too large to be protruded by the patient. The pulse, which was at first full, becomes small and quick, and the urine is much diminished or quite suppressed. The trembling limbs are cold and covered with clammy sweat, the patient passes into delirium or coma, from which he does not eventually emerge, hiccup comes on, the eyes become bloodshot, the pupils dilated, not unfrequently the motions are involuntary and of a cadaveric fœtor, the pulse becomes thready and intermittent, and death closes the scene. When the issue is to be favorable, of which the indication is copious evacuation of the bowels, the urine gradually returns, the vomiting ceases, and the patient passes into a convalescent state which often lasts for months. In many cases the disease runs its course with moderately severe symptoms, such as violent tremblings on the smallest physical exertion, and a remarkable degree of stiffness in the joints, which lasts a long time.

Morbid anatomy.—Graff succeeded only once in getting a *post-mortem* examination in this disease. The case was that of a woman who died

¹ Ref. III.

on the fourth day, severe metrorrhagia having come on; the gastric and intestinal mucous membrane was injected in places, and the intestine remarkably contracted in its lumen; there were typical signs of cerebral meningitis, the brain was soft and very hyperæmic, with much serum in the ventricles; the liver also was hyperæmic. Blood which had been drawn from a vein on the third day of the disease coagulated slowly, the clot being small, soft and gelatinous, and the serum stained red, apparently through extensive decomposition of the red corpuscles.¹

This disease occurs, as we have seen, in the very same regions where the above described disease of cattle is endemic, and it was so common in the first years of the century that several hundred persons died of it annually. From time to time it assumes an epidemic character, in correspondence with the epizootic, as in Falmouth,² Kentucky in 1854 and 1856, and most recently in 1867 in Kenton,³ Ohio, when there were fifty cases and seven deaths.

§ 81. EVIDENCE OF POISONING BY THE MILK OR MEAT.

The special interest of this disease for our inquiry lies in the question of its relation to the disease in cattle. The similarity of the symptoms as a whole in the two processes warrants us in assuming that they are connected in their causation, either in such wise that both of them proceed from one and the same cause, or that the disease in the human subject depends upon sickness in the animals, that is to say, upon a transmission from the latter to the former. While each of these theories has its supporters, by far the larger number of observers incline to the view that *the disease in man is solely the consequence of using as food the milk (and the products prepared from it) or the flesh of the diseased animals*; and hence the name of "milk-sickness" by which the disease is colloquially designated. Numerous observations by Lewis, McCall, Yandell, Crooks and others lend material support to this doctrine; not less in its favour is the fact that carnivorous mammals and birds, such as the dog, fox, wolf, buzzard,

¹ Graff explains that the anatomical condition was not perfectly ascertained for the reason that he was obliged to make the examination by candle light in the open air.

² Sutton.

³ Smith.

and vulture, which had fed on the flesh of cattle dead of the trembles, succumb. Most decisive of all, however, we may reckon the toxicological experiments which Graff made on dogs and other animals with the milk and flesh of cows that were killed by the disease; assuming that no mistakes of a gross kind have been committed, there can be hardly any doubt that these experiments have established the fact of transmission.

Graff's inquiries into the *physical, chemical and toxic properties of the flesh and milk of cows dead of the disease* have yielded the following results: In appearance and taste, the flesh differs in no respects from healthy meat; it goes putrid, however, rather sooner. Pickling it, smoking it, or treating it with various acids and alkalies did not destroy its virulent properties; somewhat prolonged boiling with galls, and subsequent careful sousing of the flesh, destroyed its toxic character in so far that only slight symptoms of poisoning ensued in the experiments on animals, after the administration of large quantities of it. Further, the poison proved to be completely insoluble in water. The milk, as well as butter and cheese made from it, showed no differences in its physical qualities (smell, taste and appearance) from that yielded by healthy cows. The following experiment by Graff affords an approximate measure of the time during which the elimination of the absorbed poison may go on in the sick animal: A cow slightly affected was brought into a shed, where she was kept perfectly quiet and properly fed; for eight days after her milk was still found to be poisonous, but in another week she was well and the milk had lost its virulent properties. The elimination of the poison ceased, accordingly, from eight to fourteen days after the seizure, and, in Graff's opinion, the *restitutio ad integrum* was not gradual but sudden.

§ 82. OTHER EXPLANATIONS OF MILK-SICKNESS. SIMILAR OBSERVATIONS AT MALTA.

Against this theory of the origin of the disease in man, a number of objections have been raised, which may be

summarised as follows : (1) Milk-sickness occurs more frequently in adults than in children, although the consumption of milk by the latter is the greater ; (2) not everyone who had drunk of the suspected milk or eaten of the suspected flesh takes the disease ; (3) cases of so-called milk-sickness have occurred where no evidence of poisoning could be adduced.

If these objections cannot be summarily dismissed, yet the trustworthiness of the observations from which they proceed is not so perfectly assured that they can invalidate the force of the facts already given. Still less can that evidence be said to be weakened by the notion which refers the cause of the disease to the *influence of malaria*—an influence assumed as affecting men and animals alike.¹ This notion, begotten of the malaria craze, rests upon the entirely erroneous assumption that the disease occurs especially on damp or marshy, that is to say, malarious soil ; and it takes so little account of the symptoms and of all the experience collected to prove the narrow limitation of the disease to particular spots, that Yandell is perfectly right in describing it as “absolutely paradoxical.”

A satisfactory solution of the whole question is only to be got, naturally, by discovering the source of poisoning, which has so persistently eluded every search hitherto. In favour of the possibility of animals conveying the poison to men, or in other words, of diseases being induced by the milk of animals (cows and goats), there are collateral observations relating to a series of cases of cholera-like sickness, which originated in all probability, if not certainly, in the same way. The older observations of that kind are those of Ollivier,² Bonorden³ and Chevalier.⁴ Of special interest is Mackay's⁵ account of the occurrence of cholera-like cases in Malta, after the consumption of poisoned milk. These cases would seem to form a slight pendant to the milk-sickness of the United States. On board a ship of war in the

¹ Sutton (as reported by Barbour). The same opinion was formerly expressed by Lea, Thompson (quoted by Haller), and Wright.

² ‘Journ. gén. de med.,’ 1827, ci, 255.

³ ‘Rust's Magazin für die Heilkde.,’ 1828, xxvii, 193.

⁴ ‘Annal. d'hyg.,’ 1846, xxxv, 138.

⁵ ‘Edinb. Med. Journ.,’ 1862, March, 825.

quarantine harbour of Valetta, eleven officers took ill suddenly with choleraic symptoms, and the one thing common to them all was that they had partaken of milk which had been brought on board; in following up the inquiry into the source and quality of the suspected milk, Mackay learned that it was a fact well known in Malta that cows which fed on a certain plant, called by the natives "tenaowta" became ill, and that their milk, when consumed by men, had the unwholesome effects above described. He was told by botanists that the poisoning of the animals was induced by a species of the Euphorbiaceæ.

CHAPTER VIII.

“ ENDEMIC COLIC.”

§ 83. DEFINITION OF THE SUBJECT.

Under such names as colic of Poitou, colic of Madrid, Devonshire colic, colica intertropica, colica vegetabilis, colique sèche, and dry bellyache,—names derived from the locality, from the supposed cause or from the most prominent symptom,—several diseases have been described in the course of the last hundred years as occurring at various parts of the globe endemically or epidemically; they correspond perfectly with lead-colic in the symptoms, although their origin from lead-poisoning was long contested, and is contested for some of them at the present day. The interest in the subject has become all the more lively from the fact that a malady has established itself in the French navy since fifty years ago, which in its development presents the symptoms of lead-colic, but in the opinion of many observers owes its origin to other causes of a climatic or miasmatic nature. The discussion of that question has recalled attention to the alleged endemic prevalence of the same form of disease in the tropics; and it has been attempted to show from these tropical observations as well *that there exists a kind of disease the same as or exactly similar to lead-colic, whose origin depends on an etiological factor other than lead-poisoning.* In order to elucidate this subject, which is as interesting historically as it is in the way of practice, I have in the first place put together in the sequel the facts ascertained as to the epidemic or endemic occurrence of colics of that kind; secondly, I have discussed the question of the “endemic colic of the tropics;” and finally, I have inquired into the subject of colic on board ship.

§ 84. HISTORICAL OUTBREAKS MOSTLY PROVED TO BE LEAD-COLIC.

The earliest reference¹ to an epidemic of what was afterwards called "*colica vegetabilis*" occurs in a sixteenth century notice by Cethæus,² according to which a severe form of colic—well-marked lead-colic, from the description of it—had been prevalent in several parts of France, Burgundy, Asturias, and Rhætia, the cause of it being ascribed to the drinking of certain strong wines, especially such as had been subjected to artificial treatment ("*ex artificiosa conditura sulphuris.*")³ Then follows the first account of "colic of Poitou" (*colica Pietonum*) by Citesius,⁴ who places the beginning of the epidemic in the year 1572; later information on this disease from the seventeenth and eighteenth centuries puts it beyond all doubt that it was an affair of poisoning by wine contaminated with lead, the same being the case with the colic mentioned by Bonté⁵ as having been prevalent in *Normandy* in the middle of last century.

Bonté distinguished two forms of the disease, the one depending on lead-poisoning and the other "*colique végétale.*" From the more recent account by Vasse it appears that lead-colic is somewhat common in Normandy where cider containing lead is drunk, the "*colique végétale*" on the other hand consisting in an intense irritation of the stomach due to perry, which has no toxic ingredients.

¹ The toxic properties of lead and the symptoms ensuing after poisoning by that metal were known to the Greek and Roman physicians, as appears from the statement in Dioscorides ('*De materia med.*,' v, cap. 103; '*De venenis*,' cap. 22, ed. Kühn, i, 769, ii, 32); Celsus (lib. v, cap. 27, § 15); Galen ('*De antidotis*,' ii, cap. 7, ed. Kühn, xiv, 144); Paulus Aegineta (lib. v, cap. 59, 62); Aetius ('*Tetrabibl.*,' iv, sermo i, cap. 45), and Actuarius ('*Method. med.*,' v, cap. 12). In speaking of colic Paulus (lib. iii, cap. 43) mentions an epidemic of it that had occurred in the first half of the seventh century, in many parts of the Roman Empire, the symptoms of which may perhaps be taken as pointing to lead-colic ("*plerisque in morbum comitiale, aliis ad artuum resolutionem, servato sensu, quibusdam ad ambo delapsus contigit*").

² In Schenck's '*Observ. med.*,' lib. iii, obs. 184, Frankf., 1600, p. 650.

³ It is undoubtedly to this disease, or to the one mentioned by Citesius, that Rivière's account of "*colicæ biliosæ species quæ in paralyisin degenerat*," relates—evidently lead-colic from the description of it. Like all his contemporaries, he finds himself quite at a loss to explain the true cause of it.

⁴ '*De novo et populari apud Pietones dolore colico-bilioso*,' Opp., Par., 1639.

⁵ '*Journ. de méd.*,' 1761, xv, 399, 1762, xvi, 300; 1764, xx, 15.

A disease perfectly the same as the colic of Poitou was observed at the time in some parts of the *Netherlands*, and in *Devonshire*. It was shown on careful inquiry that here again it was not dietetic substances of a vegetable nature that caused the disease, as had been supposed, but poisoning by lead; which fact having been recognised and the mischief removed, the disease came to an end.

It was proved by Tronchin¹ that the poisoning in Holland was due to the water-pipes having been lined with lead. In Devonshire, as Huxham² informs us, the blame was laid on the immoderate drinking of the cider for which the county is famous; until at length Baker,³ Alcock,⁴ and others brought forward proof that it was not the cider itself which was at fault, but the contamination of it with lead from the leaden vessels in which it was made.

At a more recent date the "colic of Madrid" attracted especial notice, owing to the writings on it by French physicians, who, although not denying the identity of the disease with lead-colic, were strongly opposed to the notion that lead-poisoning was at the bottom of it.

It had been stated by Hernandez⁵ and afterwards by Luzuriaga,⁶ the latter having the Academy of Medicine in Madrid with him, that the cases of colic resembling lead-colic were as a matter of fact colic due to lead, Luzuriaga pointing out that the poor in Madrid and many other parts of Spain used badly glazed vessels in which to keep their food, that the glazing, which contained lead, underwent decomposition, and that the food was contaminated by the lead thus set free. Larrey⁷ rejected this view as erroneous; he assumed instead that the extreme fluctuations of temperature common on the table-land of Castile were the true cause of the disease, a theory which was afterwards adopted by Faure,⁸ although the latter was obliged to admit the perfect similarity of the disease with lead-colic.

In this state of suspense the question remained until at length Hisern⁹ and Cuynat¹⁰ produced exact evidence of the

¹ 'De colica Pictonum,' Genev., 1757.

² 'Observ. Med.-Phys.,' Lips., 1784, iii, 54.

³ 'Essay concerning the Cause of the Endemical Colic of Devonshire,' Lond., 1767.

⁴ 'The Endemical Colic of Devonshire, &c.,' Plymouth, 1769.

⁵ 'Trat. del dolor cólico,' Madr., 1737.

⁶ 'Disert. sobre el colico de Madrid,' Madr., 1796.

⁷ 'Memoires de Chirurgie, &c.,' vol. i, Paris, 1812.

⁸ 'Des fièvr. intermitt.,' Paris, 1833, p. 409.

⁹ 'Revue mèd.,' 1840, Sept., p. 361.

¹⁰ 'Mem. de l'Acad. des sc. de Lyon,' 1843-4, p. 20.

lead-poisoning which was underlying the disease, Cuynat at the same time showing that the malady was by no means confined to Madrid or New Castile, but was found in many other parts of Spain,—in Catalonia, Andalusia, and wherever there were gross hygienic errors conducive to lead-poisoning, such as conveying drinking water (charged with carbonic acid) in leaden pipes, allowing lead to get into the wine, or keeping articles preserved with vinegar, such as capers, cucumbers and other pickles, in badly glazed vessels. Moreover, as Hisern adds, there can be at least no question of endemic colic in Madrid in recent times, inasmuch as it happened to him in an extensive private and hospital practice to see only seven or eight cases of this “Madrid colic” in the space of nine years.

The “dry bellyache” or “bilious colic” of *United States practitioners* has had the same fate as the “colique végétale,” that is to say, it has turned out to be lead-colic.

In a letter of Benjamin Franklin¹ to Vaughan, dated Philadelphia, July 31st, 1786, he says: “The first thing I remember of this kind was a general discourse in Boston when I was a boy, of a complaint from North Carolina against New England rum, that it poisoned their people, giving them the dry bellyache, with a loss of the use of their limbs. The distilleries being examined on the occasion, it was found that several of them used leaden still-heads and worms, and the physicians were of opinion that the mischief was occasioned by that use of lead. The legislature of Massachusetts thereupon passed an act, prohibiting, under severe penalties, the use of such still-heads and worms thereafter.”

The same was the case, no doubt, with the “bilious colic” that was prevalent and grew into an epidemic at Woodsborough in 1821; Staley² says of it that it perfectly resembled lead-colic, but it was not to be attributed to lead-poisoning because “there was only a small quantity of lead in the mills employed to extract the juice of the sugar canes;” the amount of lead accordingly in the rum could be but slight, and the disease was rather of a malarious nature.

Of the remarkable epidemics of “bilious colic” at New Orleans in the summers of 1849 and 1850 we have an interesting account by Fenner,³ from which it appears that the case was one of wholesale poisoning by soda-water containing lead; perhaps also the drinking-

¹ The letter is quoted in Hunter’s ‘Observations on the Disease of the Army in Jamaica,’ 2nd ed., Lond., 1796, p. 211.

² ‘Amer. Med. Recorder,’ 1873, vi, 231.

³ ‘Southern Med. Reports,’ 1850, ii, 27, 247.

water was contaminated by the lead pipes which connected the houses with the iron mains, and together represented a length of some 90,000 yards. More recently (1865) there was an outbreak of the same kind of "bilious colic" at a village in Orange County, N.Y.;¹ an investigation showed that the miller who supplied the village with flour had filled up the holes in his mill-stones with lead, the consequence being that particles of the metal got into the flour and therewith into the bread.²

§ 85. THE "DRY BELLYACHE" OF THE TROPICS.

While almost everyone has given up the belief in a disease of temperate latitudes identical in all respects with lead-colic, but in its origin dependent on other causes (such as the eating of some vegetable, or food prepared from the same, or the malarial influence); the same belief has lasted much longer, and is held by many practitioners even at the present day, as regards the endemic occurrence in the tropics of a "colique végétale" of that kind, or "colique sèche" (dry bellyache), constituting in fact a *specific tropical disease*.

The earliest information of endemics of that kind comes from the *West Indies*. The first to mention them,³ Smith⁴ and Hillary,⁵ do not touch the question of the origin of the disease; while Moseley,⁶ who recognised the similarity of the disease to lead-colic, did not admit lead-poisoning as the cause of it. On the other hand, Clark,⁷ Hunter⁸ and Turner⁹ were quite decided that "dry bellyache" was always the consequence of poisoning by lead, particularly by lead-tainted new rum;

¹ 'Account in the 'Medical News,' Philad., 1866, p. 112.

² Lead colic occurred epidemically under the same circumstances in 1858 and 1861 at two villages in the neighbourhood of Chartres, according to the accounts by Mannory and Salmon ('Gaz. méd. de Paris,' 1862, p. 208, 1865, pp. 270, 288, 302); the quantity of lead used by the miller in 1861 amounted to 20 kilos (44 lbs.); of some 400 persons who fell ill, about twenty died of the poisoning.

³ Sydenham ('De colica,' in Opp., Genev., 1736, i, 512) in speaking of colica Pictorum, remarks, "Apud insulas Caribum notissima est," although he says nothing of the cause of the disease.

⁴ 'De colica apud incolas Caribenses endemica,' Leid., 1717.

⁵ 'Observations on the Changes of the Air, and the concomitant Epidemical Diseases in Barbadoes,' 2nd ed., Lond., 1766.

⁶ 'Treatise on Tropical Diseases,' Lond., 1787.

⁷ 'Treatise on the Yellow Fever,' Lond., 1797.

⁸ L. c., 183, and 'Med. Transact.,' 1785, iii, 227.

⁹ 'Lond. Med. Gaz.,' 1832, xi, 78.

they stated, moreover, in confirmation of Chisholm,¹ that there could be no thought of a true endemic prevalence of this colic. In like manner Dutroulau,² who had, like Brassac,³ denied in his earlier papers that lead-poisoning was the cause of "colique sèche," returned subsequently to the view of Corre,⁴ who asserted, from his experiences in Martinique (and at Vera Cruz), that severe cases of colica stercoralis were common there, as they were everywhere, particularly in the tropics (for reasons to be afterwards given), but that he had never seen what is called "colique sèche" under other circumstances than as a consequence of lead-poisoning; that term, accordingly, as an "entité morbide spéciale" should be given up.

Next to the West Indies, Guiana used to be specially reputed as an important centre of endemic colique sèche, and Legond⁵ in particular, among more recent writers, has favoured that idea, rejecting altogether the notion of lead-poisoning as the cause of the disease and explaining it as a *neurosis of the sympathetic brought on by chill*. Long before him Rodschied⁶ had expressed the opinion that the existence of "dry bellyache" as an endemic was not to be thought of; colics of various kinds, he said, are without doubt somewhat common in Guiana, but among these the so-called dry colic played a comparatively small part, the disease known specially by that name having nothing whatever to do with climatic influences but depending on nothing else than poisoning by lead, particularly the lead contained in wine and rum. This explanation was afterwards accepted in every point by Hille⁷ in Surinam, Blair⁸ in British Guiana, and Lefèvre⁹ in Cayenne.

¹ 'Manual of the Climate and Diseases of Tropical Countries, &c.,' Lond., 1822, 93.

² 'Arch. gén. de méd.,' 1855, Décembre, and 'Traité des malad. des Européens dans les pays chauds,' Par., 1861, 34.

³ 'Considér. pathol. sur les pays chauds,' Montp., 1863.

⁴ 'Notes méd. rec. à la Vera Cruz, &c.,' Par., 1869, 60.

⁵ 'Gaz. des hôpit.,' 1834, March, 25; 'Journ. hebdom. de méd.,' 1835, Nr. 3, 13; 'Essai sur la névralgie du grand sympathique,' Par., 1837, and 'Revue méd.,' 1839, Mai, 239.

⁶ 'Bemerkungen über das Klima . . . von Rio Essequibo,' Frankf., 1796, 184.

⁷ Casper's 'Wochenschr. der Heilkde.,' 1842, Nr. 6.

⁸ 'Account of the last Yellow Fever Epidemic,' Lond., 1852, 21.

⁹ 'Recherches sur les causes de la colique sèche,' Par., 1859, 96, 98.

According to an account by Chapuis,¹ there was a remarkable increase of the colique sèche in Cayenne during the years 1858–60, as compared with the years immediately preceding: while the number of cases treated for that disease in 1856 and 1857 was 82 and 67 respectively, it rose in 1858 to 102 and in 1859 to 201. Of 31 patients treated for colique sèche in the first quarter of 1860, there were 6 for whom lead-poisoning was proved; for the remaining 25 no evidence was forthcoming, although the possibility of the same cause having been in operation in these cases also was by no means excluded.

For other parts of *tropical South America* we do not hear a word about any disease characterised by the symptoms of lead-colic which was not actually due to lead-poisoning. The statements of Thevenot² and Berville³ as to the frequent occurrence of colique sèche in *Senegambia* are contradicted by the later accounts of Lefèvre⁴ and Villette;⁵ most of the cases had been treated in the marine hospitals and belonged therefore to man-of-war crews; and in every case, as the authorities last named tell us, lead-poisoning could be shown to be the cause of the sickness. The same holds good, according to Monnerot⁶ and Abelin,⁷ for the disease as it occurs on the *Gaboon coast*. Nothing is known of colique sèche in Algiers, Egypt, Abyssinia, on the East Coast of Africa or in the adjacent islands; from time to time a case of severe colic comes under observation in the marine hospitals of *Mauritius* and *Réunion*, but these also can be always traced to lead-poisoning.⁸ For India and the East Indies it is the same case; nowhere in these countries is there anything heard of an endemic disease resembling lead-colic, the few cases of colique sèche which are met with at Pondicherry being almost exclusively on board French men-of-war, and most of them, as Huillet⁹ remarks, traceable to poisoning by lead.¹⁰ In the ports of *Cochin China*, as at all

¹ 'Gaz. hebdomadaire de médecine,' 1860, Nr. 36, 577.

² 'Traité des maladies des Européens dans les pays chauds,' Paris, 1840, 232.

³ 'Gaz. des hôpitaux,' 1858, 147.

⁴ L. c., 114.

⁵ 'Arch. de médecine navale,' 1866, Février, 81, Mars, 178.

⁶ 'Considérations sur les maladies endémiques observées à l'hôpital de Gabon,' Montpellier, 1868, p. 36.

⁷ 'Études sur le Gabon,' Paris, 1872, p. 29.

⁸ Lefèvre, l. c., p. 130.

⁹ 'Arch. de médecine navale,' Janvier, 1868, p. 12.

¹⁰ Smith ('Ed. Med. Journ.,' July, 1856), gives an account of an epidemic of "colic of Poitou" which was so general among the garrison stationed at Newera.

the French naval stations in the tropics, cases of colique sèche have been seen occasionally ; and here again they belonged to the crews of men-of-war, and were in every instance traceable to lead-poisoning.¹ From *China* we have information to the same effect by Lagorde,² who expresses his astonishment that anyone should have spoken of colique sèche as occurring endemically there ; during a two years' stay in Chinese ports he had seen only one case (lead-poisoning) on board a French ship-of-war, while no case of the disease among the foreign or native residents had come under his notice. Lastly, we have to give the same account of the colique sèche in *Tahiti*, where poisoning by lead in the wine could be proved for all the cases observed by Gallerand ;³ and in *New Caledonia* (Port-de-France) where occasional cases are seen on board the French men-of-war, they are always due to poisoning by lead.⁴

§ 86. ALWAYS CAUSED BY LEAD.

It follows from all these facts above adduced that the endemic or epidemic prevalence of "colica vegetabilis" or "colique sèche," in former centuries had always been an affair of wide-spread lead-colic ; that these occurrences have become less frequent as a more correct view of the origin of the malady has gained ground, and as the progress of public sanitation has diminished the chances of the disease breaking out ; that there have no doubt been epidemics of lead-colic from time to time, even within the most recent period ; but that there can be no thought at all of the disease

Ellia, in Ceylon, that in a force of 87 men there were 142 admissions including the relapses. It came out on inquiry that the epidemic was due to poisoning by lead in the sugar and in the arrack.

¹ See Richaud, 'Arch. de méd. nav.,' 1864, Mai, 351 ; Margaillan, 'Étude sur l'étiologie saturnine de la colique sèche,' Par., 1866 ; Gimelle, 'Union méd.,' 1869, Nr. 53, 694. Girard de la Barcerie ('Considér. méd. sur la Cochinchine, &c.,' Montp., 1868, 42) did not see a single case of colique sèche during a two years' residence in Cochin China.

² 'Arch. de méd. nav.,' 1864, Mars, 185.

³ *Ib.*, 1865, October, 286.

⁴ De Rochas, 'Essai sur la topogr. . . . de la Nouvelle Calédonie,' Par., 1860, 18 ; account in 'Arch. de méd. nav.,' 1866, Janv., 21.

being endemic or, more particularly, of there being an endemic “*colique sèche*” of the tropics. Almost all the cases observed in those latitudes, and described under that name, have occurred in ports among the crews of French men-of-war ; and in by far the most of these, it has been shown to be an affair of lead-poisoning. Thus the whole inquiry reduces itself practically to answering the question, *What are the circumstances of this much talked-of “colique sèche” of the French navy, or the so-called “ship colic?”*

§ 87. THE “COLIQUE SÈCHE” OF STEAMSHIPS OF THE FRENCH NAVY.

There have been occasional cases of lead colic occurring for a long time past on board ships of every flag, both of the mercantile marine and of the navy. If not in all of these, yet in most of them it has been possible to trace the cause of the sickness to a fresh coat of lead paint on board, or to beverages or preserves contaminated with lead ; sometimes the source of the poisoning has eluded all inquiry, and the disease, as Falck¹ appositely says, had a “cryptogenetic” origin assigned to it, although its cause was neither misunderstood nor denied.

Thus Buel² speaks of the somewhat frequent occurrence formerly of a disease perfectly resembling lead-colic among the engine-room staff of the steamships plying between Panama and California, remarking that the illness was probably brought on by lead in the drinking-water ; “the condensed water from the boilers,” says the report, “was at one time extensively used for drinking and culinary purposes, and a part of the process was performed in lead pipes.” Lefèvre³ communicates an interesting account of an epidemic, with the symptoms characteristic of lead-poisoning, on board an Italian merchant ship, in which the source of the poisoning was most probably the lead contained in the glazing of the utensils used for preparing the food. According to the account of Pop,⁴ there was not a single case of *colique sèche* from 1853 to 1857 in the Dutch men-of-war on the East Indian Station, and only a few cases in those stationed on the Surinam coast, and in adjoining West

¹ In Virchow's ‘Handb. der spec. Pathol.,’ ii, Abth. i, 181.

² ‘Amer. Journ. of Med. Sc.,’ 1856, April, 324.

³ ‘Gaz. méd. de Paris,’ 1861, 776, 788, 802.

⁴ ‘Nederl. Tijdschr. voor Geneesk.,’ iii, 24, 213, 217.

Indies; but in 1866 the disease became epidemic in a steamship of the fleet on the naval station at Sumatra, and they did not succeed in making out lead-poisoning to be the cause of the epidemic.¹

A remarkable contrast to this state of matters, as well as to that in the navies of England, Germany, and the United States, in which lead-poisoning, so far as we know, has never occurred on a large scale, is afforded by the *French navy*, in which "colique sèche" has played an important part since the last forty-five years, or since the time when steamships were introduced, having gone on increasing in proportion as these have displaced sailing ships in the fleet.

The disease first began to occur more frequently on board French men-of-war, as we learn from the inquiries of Lefèvre,² subsequent to the year 1840; and it was found over the whole of the West African station from St. Louis down to Cape St. Paul de Loando, on the Indo-Chinese station (Madagascar, Réunion, and the Moluccas), and on the South Sea station, its occurrence being somewhat later on the West Indian station and on board the ships on the coast of Cayenne and in the River Plate.

It is admitted on all hands that the colique sèche is absolutely the same in its phenomena as lead colic; but in

¹ Account in 'Arch. de méd. nav.,' 1867, Septbr., 169.

² 'Rech. sur les causes de la colique sèche, &c.,' Par., 1859 (the leading work). The earlier accounts are those of Dutroulau, 'Gaz. méd. de Paris,' 1851, 278; 'Arch. gén. de méd.,' 1855, Decbr., 1856, Janv.; Fonssagrives, ib., 1852, Juni, and 'De la nature et du traitement de la colique nerveuse, &c.,' Par., 1857; Marion, 'Nouv. Annal. marit.,' 1852, Aug.; Letersec, 'Observ. sur la colique nerveuse, &c.,' Montp., 1855; Petit, 'Considér. hyg. et méd. sur la col. sèche, &c.,' Par., 1855; Rochard, 'Union méd.,' 1856, Nr. 4, 5; Desjardins, 'Gaz. des hôpit.,' 1856, Nr. 16; Lecoq, ib., Nr. 5; St. Pair, ib., 1857, 340; Berville, ib., 1858, 147; Péron, 'Quelq. reflex. sur la colique sèche,' Par., 1858; Touzé, 'De la colique sèche des pays chauds,' Par., 1858; Chevallier, 'Annal. d'hyg.,' 1859, xi, 95, 296. There are more recent accounts by Lefèvre, 'Gaz. méd. de Paris,' 1861, 39, and 'Arch. de méd. nav.,' 1864, Oct., 302, Nov., 385; Luzet, 'Sur les causes et le traitem. de la c. s.,' Strasb., 1861; German, 'De la col. nerv. des pays chauds,' Par., 1862; Benoit de la Grandière, 'Relat. méd. d'une traversée de Cochinchine en France, &c.,' Par., 1862; Vidal, 'La colique sèche à la Guyane franç., &c.,' Montp., 1863; Lagarde, 'Arch. de méd. nav.,' 1864, Mars, 185; Mondot, 'Étude sur la c. s., &c.,' Montp., 1864; Richaud, 'Arch. de méd. nav.,' 1864, Mai, 351; Villette, ib., 1866, Févr., 81, Mars, 178; Follet, 'Étude sur la c. s. végétale, &c.,' Montp., 1866; Morgaillan, l. c.; Dupré, 'De la c. s. des pays chauds,' Par., 1866; Borchard, 'De l'identité de la c. s. des pays chauds et de la col. saturnine,' Par., 1866; Soboul, 'Considér. sur la c. s., &c.,' Montp., 1868; Roumieu, 'De la c. s. observée en Cochinchine, &c.,' Montp., 1869; Marnata, 'De la col. sèche, &c.,' Par., 1880.

cases where there has been no obvious occasion of lead-poisoning on board the vessels, or where the inquiries that were made have yielded no results, many observers have decidedly refused to admit the saturnine character of the disease, and have either adopted Segond's view of a neurosis of the sympathetic brought on by chill (Rochard, Marion, Chabassu,¹ Thil),² or they have pronounced for a miasmatic origin of the malady (Dutroulau),³ or even for its malarial nature (Fonssagrives, Vidal, Coste,⁴ Hervé,⁵ Roumieu). There is still another school who would admit that many cases of the ship colic are indeed cases of lead colic, but that there had been besides these a special kind of colique sèche depending on some one of the causes above mentioned. (Thil, Follet, German, Mondot.) This chaos of contradictory and quite untenable opinion has been reduced to order by Lefèvre, Director of Naval Sanitary Board at Brest; in his truly classical work, he has adduced evidence that the "*colique sèche*" of the French navy is nothing more than lead colic, and he has had the satisfaction of seeing not only old adherents of the miasmatic theory like Dutroulau⁶ come round to his side, but of gaining the unreserved adhesion of the majority of the French naval surgeons of recent years (Villette, Richand, Benoit de la Grandière, Corre, Margailan, Borchard, Dupré, and others). The regulations drawn up in consequence of this by the Ministry of Marine for the steamships of the navy, warrant us in expecting that the disease will become as rare in the French marine in future years as it was previous to 1840. In the following pages I have put together the facts which serve to explain the exceptional frequency of lead colic in the French navy, particularly in tropical waters and among certain classes of the ships' companies.

The chief stress has to be laid on the enormous amount

¹ 'Union méd.,' 1863, Nr. 126, *seq.*

² 'Remarques sur les principales malad. à la Cochinchine,' Par., 1866, 29.

³ "L'atmosphère marine des côtes," he says ('Traité,' 72), "semble être le foyer d'émergence de sa cause, qui frappe de préférence les équipages des navires en mouillage."

⁴ 'Arch. de méd. nav.,' 1867, Octbr., 299.

⁵ 'Union méd.,' 1862, Nr. 43.

⁶ In the 2nd ed. of his 'Traité,' Par., 1868, 647.

of lead used in French men-of-war with steam power, and upon certain applications of it which afford quite special opportunities for lead-poisoning. In the construction and equipment of a French war-steamer of ninety guns, there are used, according to official returns, no fewer than 13,000 kilogrammes of regulation lead, partly in the form of pipes (for conveying water), partly as receptacles, partly as plates to protect the ship's sides within, and partly as deck fastenings, the superficial area of this mass of metal amounting to upwards of 80 square metres (or about 100 square yards). We have to add to that a large quantity of oxide of lead and salts of lead for the making of putty and paint. But most important of all we have the fact that, since 1840 or since the time when the malady began to be more prevalent, the distilling apparatus supplied to the ships has been so ill designed as to have the distilled water (which is known to have a strong affinity for lead) conveyed away in leaden pipes. Another ready source of poisoning has been discovered in the so-called "charniers," or large wooden tanks for holding drinking-water, provided round their edges with mouth-pieces communicating with siphon-tubes, through which the sailors and others of the ship's company imbibe the water. These mouth-pieces are mostly made of glass; but on many of the ships the tubes themselves are made of lead, and that construction, which is open to suspicion in any case, becomes all the more dangerous from the fact that the water, whenever the ship comes into tropical latitudes, is acidulated to make it more refreshing. Lastly, it should not be omitted that the enamel of the drinking cups and cooking utensils on board French men-of-war usually contains lead; and after long use they may easily give rise to poisoning. The following facts given by Lefèvre in an open letter¹ addressed to Dutroulau will serve to show how careless the French naval authorities have been in that matter :

"Dans toutes nos possessions équatoriales l'industrie des confiseurs, des pâtisseries, restaurateurs, marchands de vins ou de comestibles, distillateurs et fabricants de sucre, s'exerce en toute liberté, sans qu'on se préoccupe du choix

¹ 'Gaz. hebdomad. de méd.,' 1860, p. 438.

des substances, qu'ils emploient, du degré de pureté des étamages et de celui des alliages des vases d'étain destinés à contenir ou à mesurer les boissons. . . Nous possédons assez de faits recueillis en France, et particulièrement en Espagne, prouvant l'altération rapide de ces vernis plombifères sous l'action des liqueurs et des aliments acides, pour n'avoir aucun doute sur la part qu'ils peuvent avoir dans la production de ces coliques, qui paraissent endémiques dans certains pays où elles ne se développent habituellement, comme vous l'avez observé aux Antilles, que sous la forme sporadique."

The greater frequency of the disease on board ships cruising in tropical waters compared with those stationed in temperate latitudes is explained without the slightest difficulty by the circumstance that a higher temperature is very materially conducive to the development of lead-poisoning. Tanquerel has shown that, of 1217 cases of lead colic, 454 occurred in summer (June—August), 309 in spring (March—May), 251 in autumn (September—November), and 203 in winter (December—February), giving a very considerable preponderance in the warmer months. In complete agreement with this is the fact that the maximum amount of the disease in the above-named regions of the temperate zone (France, England, Spain, and the United States) has always fallen in summer; perhaps also the fact that those employed in kitchens and engine-rooms, and particularly firemen, have been attacked with the colic unusually often, so much so that on board the American mail steamers it was known by the name of "fireman's colic." There is another circumstance besides, which may serve to explain the prevalence of colic in that particular class of the ship's company—I mean their having to remain constantly in confined and badly ventilated places, where the atmosphere is charged with particles of lead.

§ 88. REVIEW OF OBJECTIONS TO THE DOCTRINE OF LEAD COLIC ON BOARD STEAMSHIPS.

To all these proofs, adduced both *a priori* and *a posteriori*, of the origin of "colique sèche," the opponents of the

doctrine answer by two arguments, which require a few words of notice. In the first place they say that the disease does not occur at all on board the steamships of other navies, such as the English, American, and German, or only rarely as in the Dutch navy. This is easily explained, in my opinion, by the fact that the naval sanitary authorities of those countries have carefully avoided everything in the construction, equipment, and provisioning of their men-of-war, which might give rise to lead-poisoning among the crew;¹ whereas in the French navy there must have been something wrong. And herein lies the great merit of Lefèvre in showing what that "something" was, by means of the exact inquiries already referred to. The second objection is that in many cases of colique sèche, it has not been possible to make out any source of lead-poisoning; while, on the other hand, out of considerable numbers who were all equally exposed to the suspected influence of lead, some have become ill of colique sèche and others have escaped. These facts must be admitted; but in weighing the second argument we should keep in mind that predisposing causes play a considerable part in the etiology of lead colic, as of almost every disease. Not to mention the peculiar predisposition residing in the physiological circumstances of the individual and hardly capable of exact definition, which confers a relative immunity from the action of certain poisons or makes a peculiar liability to the same, it has been shown by Tanquerel, Galtier,² Chevalier,³ Levy,⁴ and others, that the abuse of spirituous liquors predisposes in

¹ The distilling apparatus in the French navy is mostly made of tin-plate, which is well known to contain lead. From the experiences gathered in the Prussian navy in this matter, I am able to give the following facts:—A few years ago the distilled water from a distilling apparatus which had been made at the Dantzic dockyard out of tin as an experiment, was examined, and found to contain a large amount of lead. After it had been in use for some time, the lead disappeared from the water, and it was thereupon found that the interior of the apparatus was coated with a greyish substance easily rubbed off, which was undoubtedly a deposit of lime, gypsum, &c., from the water itself. What had happened was that the lead on the surface of the tin had been dissolved out by the hot water, while the coating, although an uncertain protective, had enabled the metal to withstand the action of the water subsequently.

² 'Traité de Toxicologie,' Par., 1845, i, 659.

³ 'Annal. d'hyg.,' 1852, Oct., xlviii, 331.

⁴ 'Traité d'hyg. publ.,' ed. ii, Par., 1857, ii, 906.

an especial way to lead-poisoning ; and herein we must seek to explain the fact that just as the officers and midshipmen on board ship are much more rarely attacked than the sailors, so, when lead colic is epidemic or endemic on shore, the women and children are much less liable to it than the men. As regards the first argument, relating to the unexplained cases of colique sèche, we are quite justified in doubting whether the search after the sources of lead-poisoning had been conducted with the necessary circumspection and care ; “if one is not discouraged,” says Borchard, “by the fruitlessness of a first search, one always ends by finding the toxic metal.”

The much greater frequency of severe cases of colic, especially of colica stercoracea, in the tropics than in temperate latitudes, is explained by the disordered digestion that is so easily brought on in the former, particularly in strangers, and by the lesions of the stomach and intestine resulting therefrom. The so-called “colique sèche,” or, in other words, the disease with symptoms of lead colic, is actually nothing else than a lead colic, and we must accordingly express agreement with Le Roy de Méricourt when he says :¹

“Il n’y a pas lieu d’admettre dans le cadre nosologique, en dehors des manifestations variées et successives de l’intoxication saturnine une maladie endémique des pays chauds, donnant lieu aux mêmes symptômes se succédant dans la même manière, qui reconnaîtrait pour cause une intoxication miasmatique, tellurique ou autres. La colique endémique des pays chauds n’existe pas.”

¹ ‘Bull. de l’Acad. de méd. de Paris,’ 1876, ii, sér. v, 460.

CHAPTER IX.

ANIMAL PARASITES.

§ 89. The term "parasitic disease" includes all those forms of sickness in which the morbid cause is the residence, permanent or temporary, of living organisms within or upon the human body ; or, to express it otherwise, in which the disorder of health is brought about by animal or vegetable organisms that take up their abode for a time or for the whole of their existence in man's body and find their sustenance there, either preying upon it partly by way of abstracting matters from the tissues of their host which they appropriate to their own uses, partly by acting as mechanical irritants, or inducing disease sometimes by elaborating from materials within their reach, or by excreting from their own bodies certain chemical products which exert an injurious or toxic influence on the human organism within or upon which they reside. Of recent years few departments of medicine have grown so much as that of parasitology. Starting from the modest beginnings of the intestinal Helminthinae and a few worms and insects inhabiting the surface of the body, it has in course of time annexed the group of epiphytes and entophytes as well as a large number of parasites belonging to the lower orders of the animal kingdom which inhabit the internal organs. But there has also arisen, in the course of studying the infective diseases, a prospect of adding to this department of pathology to an extent that cannot yet be estimated, in adducing evidence that those diseases are of a parasitic nature, or that there occur in them organisms of the lowest rank of organic development—the micrococci and bacilli, so named from their form. The results hitherto arrived at concerning the part played by these parasites in the acute and chronic infec-

tions have either been given already, or remain to be given, in treating of each of the diseases in its proper place. In the following chapters my purpose is merely to deal with those diseases where the parasitism consists in the presence of more highly organised animal or vegetable forms within particular organs or parts ; and which possess further a special interest for geographical pathology, owing to their wide diffusion over the globe or to their endemicity at particular spots.

We may take it to be a general truth that parasitism is much more developed, whether we estimate it by the number of the forms under which it occurs or by the frequency of each individual form, in lower latitudes, that is to say, in tropical and sub-tropical countries, than in the temperate and cold zones.

“Les vers,” says Sigaud,¹ “occupent une grande place dans la pathologie intertropicale.” Speaking of the frequency of *Enthelminthæ* in the Nile countries, Pruner² says: “One seldom opens a cadaver in Egypt without finding specimens of one or more species.” Waring³ says: “No medical officer can have had charge even for a short time of any of the large civil dispensaries in any part of India, without having been struck with the large proportion of ‘worm cases’ which come under his observation ;” and the reports from the West Coast of Africa,⁴ Guiana,⁵ and the West Indies⁶ are to the same effect. What is here said of the *Enthelminthæ* in particular applies still more to the other parasites, the larger number of which are indigenous to the tropics.

The explanation of this is partly, no doubt, the influence which climate exerts on the variety and luxuriance of every kind of growth ; partly also the circumstances and habits of living which, as we shall see in speaking of the several forms of disease, are peculiarly favorable to the introduction of parasites into the human body. It is these habits that serve to explain the differences in the amount of parasitic disease among various races and nationalities as well as among different classes of human society.

¹ ‘Du climat et des maladies du Brésil,’ Par., 1844, 425.

² ‘Die Krankh. des Orients.,’ Erlang., 1846, 244.

³ ‘Indian Annals of Med. Sc.,’ 1859, July, 371.

⁴ Boyle, ‘Med. Account of the Western Coast of Africa,’ Lond., 1831, 402.

⁵ Rodschied, ‘Med. Bemerk. über . . . Rio Essequibo,’ Frankf., 1796, 290.

⁶ Levacher, ‘Guide méd. des Antilles,’ Par., 1840, 193 ; Thomson, ‘Edinb. Med. and Surg. Journ.,’ 1822, Jan., 43.

I. *Cestodes*.§ 90. *TÆNIA MEDIOCANELLATA*.

Among the parasites that have been longest known and are most widely distributed belong those of the tapeworm class—*Tænia mediocanellata* (*T. saginata*) and *Tænia solium*; next to these we have to consider *Bothriocephalus* and *Echinococcus* (the larva of *Tænia echinococcus* whose host is the dog).

Tænia mediocanellata has its greatest area of distribution in *Abyssinia*, where, according to the unanimous opinion of observers,¹ only a few of the natives are exempt from it, and strangers also soon become infested by it unless they are especially careful about their food and in particular avoid the favourite diet of raw meat. “Men and women, all and sundry, in this country,” says Courbon, “have the entozoon; and they take some koussou regularly every two months to prevent the more serious derangements which it might cause.” In *Egypt* and *Nubia* also, it is mostly *Tænia mediocanellata* that is met with;² and the same is true of the extensive territory, mostly occupied by a Mohammedan population, which extends from the northern coast of Africa (including *Algiers*³), to *Senegambia*,⁴ and the western *Soudan*,⁵ and, on the other side, to *Syria*⁶ and *Arabia*.⁷

¹ Aubert-Roche, ‘Annal. d’hyg.’ xxxv, 5; Hodgkin, ‘Med. Times,’ 1844, No. 266; Pruner, l. c.; Schimper, ‘Gaz. méd. de Strasb.’ 1848, Nr. 4; Bilharz, ‘Zeitschr. für wissenschaftl. Zoologie,’ 1850, iv, 35; Courbon, ‘Observ. topogr. et méd. . . sur le littoral de la mer rouge,’ Par., 1861, 35; Currie, ‘Brit. Army Reports’ for the year 1867, ix, 296; Blanc, ‘Gaz. hebdom. de méd.’ 1874, Nr. 22, 345; Rochard, ‘Bull. de l’Acad. de méd.’ 1877, 998.

² Pruner, Bilharz, ll. cc.; Vauvray, ‘Arch. de méd. nav.’ 1873, Sept., 161; Tutscheck, ‘Oest. med. Wochenschr.’ 1846, 1209.

³ Boudin, ‘Mém. de méd. milit.’ 1848, lxx, 204; Rénaud, ib., 1873, Oct., 545; Vidal, ‘Gaz. méd. de Paris,’ 1874, Nr. 22, 23; Cauvet, ib., Nr. 33; Arnould, ib., 425; Henne, ‘Mém. de méd. milit.’ 1876, Mai, 238; Rochard, l. c.

⁴ Thaly, ‘Arch. de méd. nav.’ 1867, Sept., 187; Léonard, ‘Observ. rec. au poste de Sed’ Hiou, &c.’ Par., 1869, 62; Corre, ‘Bull. gén. de thérap.’ 1877, Févr., 170; Hébert, ‘Une année méd. à Dagana,’ Par., 1880, 41; Borius, ‘Arch. de méd. nav.’ 1881, Mai, 372.

⁵ Quintin, ‘Extrait d’un voyage dans le Soudan,’ Par., 1869, 49.

⁶ Pruner, l. c.; Robertson, ‘Edinb. Med. and Surg. Journ.’ 1843, April, 247; Guys, ‘Statistique du Paschalik d’Alep,’ Marseille, 1853, 63; Rochard, l. c.

⁷ Pruner, Courbon, ll. cc.

Of the frequency of the parasite in *Algiers* we are sufficiently assured in the account by Boudin; during the years 1840—48 sixty-eight cases of it occurred in an average strength of 100,000 men, whereas among 250,000 (average) men in France during the same period there were only seven cases, so that the parasite was twenty-three times commoner in *Algiers* than in the mother country. Subsequent observers (referred to below in the paragraph relating to France) have called attention to the increasing frequency of *Tænia mediocanellata* in the latter since Algerian cattle began to be imported. With reference to *Senegambia*, Léonard has expressed his astonishment that *tænia* should be so remarkably common there, inasmuch as the natives, being Mohammedans, eat no flesh of swine; but this mystery has been cleared up by Corre, who points out that raw beef is a favorite article of diet with them, and that the tapeworm is not *Tænia solium* but *T. mediocanellata*. It would appear from the statements of Quintin that the circumstances are the same in the *Soudan*, where pork is never used, but where beef dried in the sun serves as the food of the negro population. The chief seats of the parasite in *Syria*, according to Pruner and Guys, are the mountainous regions, and the vicinity of Aleppo; and in *Arabia*, according to Pruner, the plateau of Assir. In Jeddah, on the Arabian shore of the Red Sea, it is said by Courbon to be of rare occurrence.

A notice by Bérenger-Feraud¹ throws light on its prevalence in *Senegambia*; among 159 patients admitted for tapeworm into the naval hospitals of Toulon and Cherbourg, the only worm found was *Tænia inermis*, and of the whole number 102 had acquired the parasite in *Senegambia*. The accounts from various parts of the *West Coast of Africa*² all bear witness to the very frequent occurrence of the tapeworm disease there, and although the species of the parasite is not stated with precision, there can be no doubt that it is mostly *Tænia mediocanellata*. This conjecture is made all the more probable by the fact that the same species of *tænia* occurs in *South Africa* (particularly the *Cape*³); so

¹ 'Bull. gén. de thérap.,' 1882, 15 Aug., 97.

² Boyle, l. c.; Daniell, 'Sketches of the Med. Topogr. . . of the Gulf of Guinea, Lond., 1849, 53; Moreira, 'Jornal das sc. med. de Lisboa,' xv, 121.

³ Hodgkin, l. c.; Black, 'Edinb. Med. and Surg. Journ.,' 1853, April, 262;

that the African continent would appear to be its habitat in general. The occurrence of tapeworms among Hottentots who had served in the war in Caffirland is mentioned by Scherzer; and it would appear that not *Tænia solium* but *T. mediocanellata* is meant here, from the fact that the tribes in question avoid the flesh of the pig while they esteem raw beef as a delicacy.

For *India*, the available data¹ seem to show that tapeworm is indigenous principally in certain provinces of Upper India belonging to the Bombay Presidency and the N. W. Provinces, especially in the Deccan and the Punjaub, and to a less extent in Lower Bengal, Madras, and the southern districts; but in that country the Hindu race, as we shall see presently, enjoys an almost complete immunity from the parasite, and it is practically the European residents and the flesh-eating Mohammedans who suffer from it. So far as concerns the Mohammedan population we are justified in concluding that it is the *Tænia mediocanellata* which is found among them; and the same would appear to be the case for the most part with the Europeans living in India, and particularly for the British troops in the Punjaub, among whom tapeworm has been very often observed of late. The use of uncooked flesh, including even beef, by the troops in India had already been indicated by Hoile as the cause of the tapeworm so often found among them. In a more recent paper² attention is drawn to the increase of the disease especially in the Bombay Presidency and in the Punjaub; and in a subsequent notice relating to the same fact it is stated that "the prevalence of tapeworm in the regiments in the Punjaub has been recently shown to depend upon the consumption of beef infected with the *Tænia mediocanellata*."³

The statement of Bernard,⁴ that *Tænia solium* occurs in

Scherzer, 'Zeitschr. der Wiener Aerzte, 1858, 152; Adams, 'Med. Times and Gaz.,' 1859, Dec., 549.

¹ Anderson, 'Ind. Annals of Med. Sc.,' 1852, Oct., 87; Hoile, *ib.*, 1857, April, 457; Gordon, 'Med. Times and Gaz.,' 1856, Nov., 512; 1857, May, 429; Adams, *l. c.*; Chipperfield, 'Madras Quart. Journ. of Med. Sc.,' 1861, Jan., 78; Waring, *l. c.*

² 'Med. Times and Gaz.,' 1867, Nov., 573.

³ 'Lancet,' 1868, Jan., 59.

⁴ 'De l'influence du climat de la Cochinchine, &c.,' Montpell., 1867, 50.

China and *Cochin China* in consequence of the use of measly pork, is confirmed by Beaufile;¹ but the latter agrees with Rochard that *Tænia mediocanellata* is much the more common form.² The same is true also of *Japan*³ and other parts of Northern Asia, as well as of the Mongolian race of Barjutes inhabiting the country around *Lake Baikal*.⁴

Of the somewhat frequent occurrence of *Tænia mediocanellata* in Europe,⁵ we have evidence from *North Germany*⁶ (East Prussia, Berlin,⁷ Leipzig, according to Wagner, and a few districts of Thuringia⁸), as well as from Würtemberg⁹ and Vienna. In Copenhagen, *T. mediocanellata*, as compared with *T. solium*, has become a good deal more common of recent years (whereof more in the sequel); so that while the proportion of the former to the latter was 53 to 37 down to 1869, it came to be in the ratio of 66 to 19 in the years from 1869 to 1880.¹⁰ In *England*, where attention was long since drawn to the numerous cases of *tænia* among fleshers who were known as “beef eaters” *par excellence*, we learn from Welch¹¹ that *T. mediocanellata* is very common. So it is also in the *Netherlands*, while in *Belgium*¹² the *Tænia solium* is distributed about equally with it. In *Switzerland*¹³ *T. mediocanellata* appears to be a good deal more prevalent than *T. solium*; of 199 patients with *tænia*,

¹ ‘Arch. de méd. nav.’ 1882, Avril, 265.

² Béranger-Féraud (l. c.) found nothing but *Tænia inermis* among 44 patients invalided home from *Cochin China* with tapeworm, who were admitted into the naval hospitals of Toulon and Cherbourg.

³ Wernich, ‘Deutsche med. Wochenschr.’ 1878, Nr. 6; Leuckart, ‘Parasiten,’ 2 Aufl., i, 605, on the authority of Prof. Bälz.

⁴ Kaschin, ‘Petersb. med. Zeitschr.’ 1861, Dec. Almost the whole of a division (500), composed of Barjutes, and quartered in Irkutsk, suffered from *tenia*. In 180 of them, who died of other diseases and were examined *post mortem*, he failed to find tapeworms only twice.

⁵ Compare Knoch, ‘Berl. klin. Wochenschr.’ 1864, Nr. 30 ff.

⁶ Virchow’s ‘Archiv für pathol. Anat.’ 1857, xi, 80.

⁷ Robinski, ‘Berl. klin. Wochenschr.’ 1874, Nr. 37.

⁸ v. Conta, ‘Zeitschr. für Epidermiol.’ 1871, Nr. 10, 11. Among 18 patients with tapeworm at Jena, Gerhardt found *T. mediocanellata* in 15 and *T. solium* in 3.

⁹ Weishaar, Knoch, l. c.

¹⁰ Krabbe, ‘Ugeskr. for Læger,’ 1869, xxiii, Nr. 8 and 1880, N. R., xii, Nr. 23.

¹¹ ‘Journ. of Microscop. Science,’ 1875, Jan.

¹² Knoch, l. c.

¹³ Zaeslein, ‘Correspondenzbl. für Schweizer Aerzte,’ 1881, Nr. 21.

180 had *T. mediocanellata* and only 19 *T. solium*. Indeed, the former species seems to have been found in recent times much more often than it used to be; and in *France* also, a considerable increase in the cases of *T. mediocanellata* has been noted of late,¹ of which fact an explanation will be given in the sequel. For *Italy* I find only two notices relating to the question—by Grasse² and Marchi; according to these 16 out of 19 cases in Milan were *T. mediocanellata*, while in Florence the proportion of that species to *T. solium* was 34 to 1.

In the medico-topographical accounts from the *Western Hemisphere*, there are on the whole very few references to the occurrence of tapeworm, and most of them either relate to *T. solium* or leave the species undetermined. In Mantegazza's³ account of the truly endemic prevalence of tapeworm in the *Argentine Republic*, and especially in *Entre Rios*, we read that an explanation should be looked for in the great liking of the people for partially cooked beef,—the beef-steaks à la Tartare; so that we may fairly take the parasite in that case to be *T. mediocanellata*.

§ 91. TÆNIA SOLIUM.

The area of distribution of *Tænia solium* extends in like manner over the greater part of the globe, or wherever swine's flesh is used. But that species has been met with much more rarely than *T. mediocanellata*, particularly of recent times. Moreover many of the earlier notices of *T. solium* are based upon errors of diagnosis⁴ and relate in reality to the other species.

¹ Vidal, Rochard, ll. cc.; Decroix, 'Abeille Med.,' 1876, Juin; Bérenger-Féraud, l. c.

² 'Gaz. med. Lombard.,' 1879, No. 12.

³ 'Lettre med. sulla America meridionale,' Milano, 1860, i, 100, 160.

⁴ The derivation of the adjective "*solium*," which occurs first in the medical writers of the middle ages, remains a question. At all events, it has nothing to do either with *solium*, "the throne," and still less with *solus*, "alone." Professor Krehl, the Orientalist, conjectures (as we learn from Leuckart, 'Parasiten,' 2nd ed., i, 519) that it is a corruption of the Syriac word "*schuschl é*," meaning "chains" (as in tape-worm or chain-worm), and that from it had come the Arabic word "*susl*" or "*sosl*," well known to the mediæval physicians. It was the mis-

The widest prevalence of *T. armata* occurs in the interior governments of *Russia*, such as Moscow,¹ as well as in *East Prussia*, some parts of *Thuringia*,² of *Belgium*³ and of *Switzerland*,⁴ in *Roumania*⁵ and in *Turkey* (but only to a small extent and naturally only among those of the population who do not belong to the strictest of Moslems).⁶ In the Western Hemisphere, it occurs in *Newfoundland* where Gras⁷ assigns the cause of it especially to the use of pork; it is found also in Mexico, where, as we learn from Semeleder,⁸ pork is one of the chief articles of food. It is impossible to make out which of the species of *tænia* it is that occurs so often in *Brazil*, especially among the negroes, Sigaud's⁹ account making mention of it merely as "ver solitaire."

Lastly, I shall mention a few regions which have a very remarkable immunity from tapeworm of whatever species. Among them is *Iceland*, where, as Finsen¹⁰ tells us, there is a dislike of pork among the inhabitants, and where flesh of any kind plays a very subordinate part among the articles of food. Another of these regions is *Greenland* (particularly South Greenland), where tapeworm does not occur at all, so far as Lange's¹¹ information goes. In *Guatemala*, according to Bernoulli,¹² tapeworm is of rare occurrence; and in

taken idea of *solium* being derived from "solus," and of its denoting the occasional occurrence of a single parasite in an individual's intestine, that unquestionably led to errors of diagnosis, which were all the more likely to occur for the reason that practitioners were not generally acquainted with *T. saginata* until quite recent times. In those cases where only one parasite was found in an individual, it was thought necessary to assign it to the species of *T. armata*. Leuckart points out, as Davaine had done before him, that it is really *T. medio-canellata* which seldom occurs in more than a single specimen in a person's intestine, whilst *T. solium* is mostly found in numbers.

¹ Knoch.

² v. Conta, l. c.

³ Knoch, l. c.

⁴ Zaeslein, l. c.

⁵ Leconte, 'Considér. sur la pathol. des provinces du Bas-Danube,' Montp., 1869, p. 49. He is explicit in saying that he had not seen a single case of illness from tapeworm among the numerous Moslems and Jews in Roumania; pork being cheap, the consumption of it was extensive.

⁶ Rigler, 'Die Turkei und deren Bewohner, &c.,' Wien, 1852, ii, 209.

⁷ 'Quelques mots sur Miquelon,' Montp., 1867.

⁸ 'Wien. med. Presse,' 1873, Nr. 34.

⁹ L. c., 133, 425.

¹⁰ 'Jagttagelser angaaende sygdomsforholdene i Island,' Kjöbenh., 1874, 108.

¹¹ 'Bemaerkn. om Grönlands Sygdomsforhold,' Kjöbenh., 1864, 43.

¹² 'Schweiz. med. Zeitschr.,' 1862, iii, 100.

Martinique it did not happen to Ruz¹, during a practice of many years among creoles and whites, to see a single case of *tænia*.

§ 92. DISTRIBUTION OF *T. MEDIOCANELLATA* AND *T. SOLIUM*
CORRESPONDS WITH A POPULAR DIET OF BEEF AND PORK
RESPECTIVELY.

The peculiarities in the geographical distribution of the two species of *tænia*, the greater frequency of the parasites in one region than another, the differences in their occurrence among the various *races and nationalities*, the increase or decrease of one or other species from time to time in one and the same place,—all these things are explained without difficulty when we take account of the well-established fact that *Tænia mediocanellata* develops from the cysticercus that occurs in cattle, and *Tænia solium* from a larva infesting the pig, the occurrence of the one species or the other in man depending accordingly upon the access of the embryos of one bladder-worm or the other to the human intestine.

The somewhat rare occurrence of *T. armata* compared with *T. saginata* in Asia and Africa depends essentially on the fact that the use of pork is much restricted, either on religious grounds, as among orthodox Mohammedans, or from prejudice against it, as in Abyssinia and among the natives of South Africa (as well as in Iceland); and that it is mostly among the European residents that it obtains. Again, the immunity enjoyed by particular races, such as the Egyptians, the Hindus, the Malays and others, is a consequence of their almost exclusive vegetable diet, an infection by cysticercus being a rare occurrence.

In India, says Chipperfield, tapeworm is, on the whole, rare among nationalities other than the European. Waring's opinion, though less absolute, is to the same effect: "Tapeworm is confined almost exclusively to the flesh-eating Mussulman or the omnivorous European soldier." In Travancore, a purely Hindu State, he had not seen a single case of *tænia* during a six years' residence. Dr. Sperschneider, who served eight years as surgeon to the Nair brigade (a division of 1800 men composed entirely of pure-caste Nairs or Sudras), had likewise seen no case of tapeworm; and the official returns from the Travancore Circar

¹ 'Arch. de méd. nav.,' 1869, Juin, 440.

during twenty years are to the same effect. Of 95 cases of *tænia* which Anderson observed in Upper India, 86 occurred in European soldiers, 8 in Moslems, and a single case in a Hindu, belonging to one of the lower castes which did not restrict itself to a purely vegetable diet. Huillet¹ also states that in Pondicherry he had seen tapeworms only in Europeans.

We have already adverted more than once to the fact that the tapeworm parasites, and for obvious reasons *T. mediocanellata* in particular, are especially frequent in those countries where flesh in the raw or half-cooked state is a favourite article of food. In this connexion we may note the fact brought forward by the French observers above mentioned, that *Tænia mediocanellata* has become considerably more common in France since the abundant importation of beef from Algiers and the use of the same in the raw state as an easily digested and strengthening article of food for patients and convalescents. In like manner Krabbe for Denmark and Zaeslein for Switzerland assign the increased number of cases of tapeworm to the same cause. Lastly, we have to remark that several recent observers (Krabbe, Küchenmeister,² and others) have ascertained that there has been a considerable decrease of *T. solium* dating from the time when the general outbreak of trichinosis in man either led to a restricted use of uncooked pork on account of the danger of infection from it, or brought protection to the public against measly pork through the system of inspection.

§ 93. *BOTHRIOCEPHALUS LATUS*.

THE distribution area of *Bothriocephalus latus* is very much smaller than that of *T. saginata* and *T. solium*. It appears, indeed, so far as we are warranted in concluding from the facts before us, to be confined practically to a few spots in Europe, such as the coast-regions of Sweden and Finland, the Baltic provinces of Russia, including St. Petersburg, a few districts in the east of Russia, and the western cantons of Switzerland with the adjoining departments of France.

¹ 'Arch. de méd. nav.,' 1868, Févr., p. 87.

² 'Die Parasiten des Menschen,' 2 Aufl., Leipz., p. 94.

In Sweden it is truly endemic on the coast of the Gulf of Bothnia, in Norrbotten, Westerbotten, Westernorrland and Gefleborg-Län;¹ it is rarer on the Baltic coast, but it has been seen in Blekings-Län.² The district most affected is Norrbotten-Län, where the number of those suffering from the parasite is estimated at half the population; in Haparanda there is said to be hardly a household in which one or more persons are not the subjects of it. In *Finland*³ also, the bothriocephalus occurs mostly on the coast of the Gulf of Bothnia. Of its prevalence in the Baltic provinces, we have information from Moritz,⁴ Erdmann,⁵ Knoch,⁶ (who also mentions it as occurring in the eastern parts of Russia), Böttcher⁷ and Braun.⁸ In St. Petersburg according to an estimate by Attenhofer⁹ in 1817, about 15 per cent. of the population suffered from it. In *Switzerland*, according to the very careful inquiries of Zaeslein (l. c.) bothriocephalus is endemic (and remarkably common) only on the shores of the lakes of Bienne, Morat, Neuchatel, and Geneva; in these localities, according to Zaeslein, the parasite occurs with a frequency that can only be compared to the general prevalence of *T. saginata* in Abyssinia; whereas only occasional cases of bothriocephalus are met with on the shores of all the other Swiss lakes.

In *Poland* (according to Knoch), in *East Prussia* and on the Pomeranian coast, in *Denmark*,¹⁰ in *Holland* and *Belgium*,¹¹ and about the *Italian Lakes* (Knoch) we find this parasite in considerable frequency, though much more rarely than in the countries before mentioned. Finally, bothrio-

¹ Huss, 'Om Sverges endem. sjukdomar,' Stockh., 1852, 2; Berg, 'Bidrag til Sveriges med. Topogr. och Statistik,' ib., 1853, 10, 16, 23, 36, 184; Wistrand, 'Öfversigt af helso-och sjukvården i Sverige 1851-60,' Stockh., 1863, 5.

² 'Medicinal-Berättelse för år 1866,' 22.

³ Wistrand.

⁴ 'Spec. topogr. med. Dorpatensis,' Dorpat, 1823.

⁵ 'Dresdener Zeitschr. für Natur- und Heilkde.,' 1827, v, 160.

⁶ 'Berliner klin. Wochenschr.,' 1864, Nr. 30 ff.

⁷ In 'Virchow's Arch.,' 1864, Bd. 30, 97.

⁸ Ib., 1882, Bd. 88, 119, and 'Petersb. med. Wochenschr.,' 1882, Nr. 16.

⁹ 'Med. Topogr. der Hauptstadt St. Petersburg,' Zürich, 1817, 226.

¹⁰ Krabbe, ll. cc.

¹¹ Boudin, 'Géogr. et statist. méd.,' i, 337; de Mattos and Israël, 'N. Arch. voor Geneesk.,' iii, 26; Knoch, l. c.

cephalus occurs in more casual cases at various points of *North* and *South Germany*—at Hamburg and Berlin, in South Bavaria,¹ at Heilbronn,² Ulm,³ Biberach,⁴ and other towns of Würtemberg,—as well as in *Brittany*⁵ and *Ireland*.⁶ In the regions beyond Europe, it would appear to be very rare, or at all events there are no sufficiently trustworthy records of it. According to a statement of Balfour's,⁷ bothriocephalus frequently occurs in children sent home from *Ceylon* to England, and Pop⁸ speaks of it being found among the crews of ships-of-war on the *Dutch East Indian station*. The accounts⁹ of bothriocephalus in *South Africa* rest most probably on errors of diagnosis, the more likely species being *Tænia lata* (or *saginata*). The description¹⁰ of a case of the bothriocephalus in an English officer in *Canada* is more trustworthy; and we may credit also Semeleder's¹¹ statement that the same species occurs along with *Tænia solium* in *Mexico*.

§ 94. BOTHRIOCEPHALUS ASSOCIATED WITH THE SHORES OF SEAS OR LAKES.

A glance at the distribution-area of bothriocephalus will show us that it is mostly *indigenous to the sea-coast and to the shores of lakes and other inland waters*. The Swedish practitioners are clear in their statements that it is almost exclusively the inhabitants of the coast who suffer from it, those dwelling even a few miles inland being nearly exempt. Zaeslein, also, has shown with much exactness that the same holds good for Switzerland. In that country he distinguishes

¹ Bollinger, 'Bayr. ärztl. Intelligenzblatt,' 1879, Nr. 15, 155.

² Betz, 'Württemb. med. Correspondenzbl.,' 1850, xxx, 262.

³ Majer, *ib.*, vi, 192.

⁴ Hofer, *ib.*, viii, 308.

⁵ Boueix, 'Journ. de méd.,' lxxv, 415.

⁶ Frazer, 'Dubl. Quart. Journ.,' 1868, Nov., 324.

⁷ In Boudin, l. c.

⁸ 'Nederl. Tijdschr. voor Geneesk.,' 1859, iii, 26.

⁹ Scherzer, Fritsch, ll. cc.

¹⁰ Leared, 'Brit. Med. Journ.,' 1874, May, 649.

¹¹ 'Wien. med. Presse,' 1873, No. 34.

four zones of frequency. The first of these, embracing the villages lying close down to the lakes, is to be regarded as the proper area of infection. In the second, comprising the country one to four leagues inland from the lakes, the parasite is much rarer; it no longer occurs, as in the first zone, among all classes equally, but more frequently among the industrial part of the population, and less so among the agricultural, the inhabitants of that zone becoming infected for the most part not at home but during their visits to the lake shore. As regards the third zone, comprising the towns, large and small, at a distance of more than five leagues from a lake, the infection may in most cases be traced with certainty, or at least with probability, to the French Alpine departments, although there are a few places such as Burgdorf and Thun for which the autochthonous occurrence of the parasite cannot be altogether denied. Finally, in the fourth zone, distant from the lakes more than six leagues, bothriocephalus is met with either in mere occasional cases, or not at all.

This narrow delimitation of the area of bothriocephalus to sea-coasts and the shores of lakes and rivers has given rise to the suggestion that its occurrence is closely connected with a *fish diet*; but it is only lately that the inquiries of Braun have furnished definite evidence on the general question as well as on the mode of invasion of the parasite. In the first place he had an opinion to controvert which Knoch¹ had given currency to, that the eggs and embryos of bothriocephalus develop at once into the worms without an intermediate larval stage. He then proceeded to show by experiments on cats, that "the asexual bothriocephali found in various organs and tissues (such as the muscles) of the pike and quab, are the immature forms of the *Bothriocephalus latus* of man, and that these fishes are to be regarded as the intermediate hosts and the sources of infection."

¹ In 'Virchow's Archiv,' 1862, Bd. 24, S. 453.

§ 95. ECHINOCOCCUS.

We have accounts of the prevalence of *echinococcus* from many regions of the *Eastern Hemisphere*, but unfortunately these are not such as to afford the means of estimating the relative frequency of that parasite at the various points within its distribution area. In the form of an endemic malady, it occurs, so far as we know at present, in only two countries—in *Iceland* and in the Australian colony of *Victoria*.

Our first trustworthy information as to the endemic of echinococcus in *Iceland* we owe to Schleisner;¹ although it follows from the references of earlier practitioners to “infarctus,” “obstructio hepatis” and “heptalgia,” that the history of the disease in that country goes back to remote times. It was Schleisner, however, who first showed that this disease of the liver depended on the presence of a parasite within the organ, that the same parasite occurs in other parts of the body, and that the malady has a most injurious influence upon the health and working capacity of the population. His observations have been subsequently confirmed in part, and in part extended by Eschricht,² Krappe,³ Hjaltelin,⁴ Finsen,⁵ and Galliot.⁶ Among 327 patients that were under his treatment, Schleisner found echinococcus fifty-seven times, or in 18 per cent. of the cases; and he estimates from the twenty years’ observations of Thorstensen, that the parasite infests one-seventh of the whole population of Iceland. Eschricht and Hjaltelin consider that estimate as not excessive, while Krabbe and Finsen would put it much lower. Finsen, whose experiences relate to the northern districts and who does not deny that echinococcus is more common in the south, would make it one-fortieth of the inhabitants, and Galliot thinks that one-

¹ ‘Island undersøgt, &c.,’ Kjöbenh., 1849, 4—16.

² ‘Undersøgelser over den i Island endemiske hydatidesygdom,’ Kjöbenh., 1853.

³ ‘Helminthol. undersøgelser i Danmark og paa Island, &c.,’ ib., 1865.

⁴ ‘Edinb. Med. Journ.,’ 1867, Aug., 137, and in ‘Dobell’s Reports,’ 1870, 286.

⁵ ‘Ugeskrift for Læger,’ 1867, iii, Nr. 5—8, and ‘Jagttagelser, &c.,’ Kjöbenh., 1874, 65.

⁶ ‘Bull. gén. de thérap.,’ 1879, Aug., 97.

thirtieth is not too high. It is a noteworthy fact that here as elsewhere the female sex suffers to a much greater extent than the male. Among 385 cases with echinococcus Schleisner found 212 in women, and Finsen had 132 female patients in a total of 189.

The first accounts¹ from *Victoria* date from the year 1863; the number of cases increased so quickly that even in 1867 Richardson² spoke of the hydatid disease as "exceedingly common," adding that "the affection has become so prevalent of late years that it may be called a disease of the country," and that it was equally common in towns and in rural districts. These statements are confirmed by later authorities,³ among others by Thomas who gives us a means of estimating the frequency of the malady approximately when he tells us that 307 deaths from echinococcus were officially registered during the ten years from 1867 to 1877 in a population of about 800,000.

Besides those two endemic centres, we have accounts of the somewhat frequent occurrence of the parasite in *India*, a certain proportion of the endemic hepatic abscesses of that country, as Cleghorn⁴ observes, being referable to it. Echinococcus is found not unfrequently also in *Algiers*,⁵ *Egypt*,⁶ and *Russia*,⁷ as well as in *England* and *France*.⁸ Of 22 cases of hydatids of the liver in *Switzerland*, 13 occurred in the north-east of the country, 3 in Basel, 3 in Neuchatel, 2 in Geneva, and 1 in Bern.⁹ From many parts of *Germany*, also, such as Dresden, Rostock, Berlin, Breslau, Jena, &c., we learn¹⁰ that echinococcus is not unfrequent; but these data relate only to

¹ Hudson, 'Austral. Med. Journ.,' 1860, April; Ralph, *ib.*, July; Sutherland, 'Victoria Med. Record,' 1863, Feb.

² 'Edinb. Med. Journ.,' 1867, Dec., 529.

³ Notice in 'Brit. Med. Journ.,' 1871, Dec., 783; Bird, 'Med. Times and Gaz.,' 1873, Aug., 164; Thomas, 'Lancet,' 1879, March, 297.

⁴ 'Indian Med. Gaz.,' 1871, March.

⁵ Vital, 'Gaz. méd. de Paris,' 1874, Nr. 22, 23.

⁶ Bilharz, 'Zeitschr. für wissenschaftl. Zoologie,' 1853, iv, 53; 'Zeitschr. der Wiener Aerzte,' 1858, 447.

⁷ Knoch, 'Petersb. med. Zeitschr.,' 1866, x, 245.

⁸ Cobbald, 'Lancet,' 1875, June, 850.

⁹ Zaeslein, *l. c.*, 681.

¹⁰ The references down to 1877 have been collected by Neisser ('Die Echinococcen-Krankheit,' Berl., 1877, 34).

hospital practice and are available neither for comparison among themselves nor for estimating the proportion of cases in the respective populations. In the *Western Hemisphere* it would appear to be rare ; at all events Osler,¹ after searching carefully in museum catalogues, journals and proceedings of societies, has found only 61 cases for the whole of North America. Of these Montreal had 3 cases (in 800 sections), and of the whole 61, it was probable that many had occurred in immigrants from other countries.

§ 96. ECHINOCOCCUS THE LARVA OF A TÆNIA INFESTING THE DOG.

The presence of echinococcus in man (and the larger animals) is due, as is well known, to the introduction of the eggs of a tapeworm which infests the dog, the *Tænia echinococcus* ; the frequency of hydatids among a population would be in proportion, therefore, to the number of the dogs, and to the intimacy of the relations between those animals and their masters' households, or to the careless way in which the members of a family keep company with the dogs. We may thus explain the prevalence of the parasite among the pastoral populations of Iceland and the Australian colony. The number of dogs kept in Iceland is proportionately much larger than anywhere else, and the enormous frequency of echinococcus among the cattle on the island, and even more among the sheep—Hjaltelin says that one-fifth of all the grown sheep have the parasite—affords abundant opportunities for the shepherds' dogs to be infected with the bladder-worm. The Iceland cowherds have notoriously little sense of cleanliness, and they are all the more ready to share with their highly treasured dogs not only their dwellings but their platters also, and even their beds, because the risk of infection from the dog is a thing unknown to them. Furthermore, the dogs have free access to the store-rooms, whose contents they be foul with their dejecta² ; and it can only be because a few

¹ 'Amer. Journ. of Med. Sc.,' 1882, Oct., 475.

² The assertion of one writer, that quacks in Iceland use the excrement and urine of the dog as a medicine for internal administration, is declared by Finsen to be a fable.

out of the many thousands of *tænia*-eggs entering the human intestine ever reach the mature stage of bladder-worms, that a very much large number of persons in that country do not suffer from echinococcus. Finsen is explicit in stating that the parasite is hardly met with among the more civilised of the Icelandic population, among families occupying commodious houses and duly practising cleanliness. It is unquestionably the same circumstances that explain the prevalence of echinococcus in Australia, where the population is mostly engaged in cattle rearing, and where the sheep, as Hudson informs us, suffer from echinococcus to the extent of at least 4 or 5 per cent. The first cases treated in the Melbourne Hospital, according to Richardson, were shepherds. "It does not require much imagination," he says, "to follow the course of these embryonic tapeworms eaten by the shepherd's dog. They are matured in the dog, passed as tapeworms over the pasturage of other sheep, the ova are again taken into the stomach and system of the sheep, and circles of propagation are established." It would be of great interest to discover what is the state of matters as regards this disease among other pastoral peoples living, like those spoken of above, in a half civilised state; and at the same time to obtain more accurate information on the geographical distribution of the *Tænia echinococcus* itself.

II. *Trematodes*.

§ 97. DISTOMA HÆMATOBIUM. ENDEMIC HÆMATURIA OF EGYPT AND SOUTH AFRICA.

The parasitic trematode which was first described by Bilharz¹ and Griesinger² and named by them *Distoma hæmatobium*, has been found hitherto as an endemic cause of sickness at only two points of the globe, both on the African continent, namely, *Egypt* and the *Cape of Good Hope*. From the former

¹ 'Zeitschr. für wissenschaftl. Zoologie,' 1852, iv, 59; 'Wien. med. Wochenschr.,' 1856, Nr. 4, 5, and 'Zeitschr. der Wiener Aerzte,' 1858, 447.

² 'Arch. für. physiol. Heilkde.,' 1854, xiii, 561.

of these we have accounts by the two observers already named and by Simpson,¹ Sonsino,² and Damaschino³; and for the latter territory the occurrence of the parasite is described by Harley,⁴ Fritsch,⁵ and Batho.⁶ It is impossible to decide, for the present, whether the *endemic hæmaturia* of the *more central parts of Africa* is associated with these parasites, as some travellers⁷ state, or, as seems to me to be more probable, with *Filaria sanguinis*.⁸

In *Egypt* the habitat of the parasite is strictly limited to the coast and to the banks of the Nile within the Delta; and in those localities it is enormously frequent. Griesinger found it in 117 out of 363 bodies, but he believed that the minor degrees of the malady, and particularly the beginnings of it, had been overlooked by him many times. And Sonsino, who found it in 13 out of 31 bodies, estimates the number of cases of distoma at a much higher ratio even than that. At the *Cape* its frequency is equally great; there also it is strictly confined to the coast territory and to the banks of a few streams for a distance of some ten or twenty miles up from the sea. Its principal seats are the south-eastern districts of *Cape Colony* about Algoa Bay, particularly Uitenhage and Port Elizabeth; the neighbourhood of King William's town and East London in *Caffraria*; and several places in *Natal*, on the banks of the Umlasi, the Ungeni (from Port Natal up to Pietermaritzburg), and the Umhloti (Verulam).

¹ 'Brit. Med. Journ.,' 1872, Sept., 320.

² 'Ricerche intorno alla Bilharzia Haematobia, &c.,' Cairo, 1874, and 'Arch. gén. de méd.,' 1876, Juin, 652.

³ 'Gaz. hebd. de méd.,' 1882, 365.

⁴ 'Med.-Chir. Transact.,' 1864, xlvii, 55; 1865, xlviii, 161; 1869, lii, 379; 1871, liv, 47.

⁵ 'Arch. für Anatomie,' 1867, 752.

⁶ 'Brit. Army Med. Reports,' 1870, xii, 502.

⁷ Allen ('Lancet,' 1882, July, p. 51) is of opinion that all the rivers of Africa, from Egypt to the Cape, contain the parasites.

⁸ See the section on *Filaria sang. hominis*.

§ 98. MODE OF ACCESS OF DISTOMA HÆMATOBIUM
TO THE HUMAN BODY.

Within the human body, as is well known, the parasite is found mostly in the blood of the portal system of veins—in the mesenteric veins—and in the vessels of the urinary bladder. From the latter it reaches the mucous membrane of the bladder, sometimes also the ureters, and even the pelvis of the kidneys. In these situations it sets up serious local lesions, leading to blood in the urine (one of the forms of *hæmaturia intertropicalis*), the formation of *calculus* (uratic and phosphatic deposit around clusters of the parasite's eggs), the endemic prevalence of which in Egypt is essentially due to *Distoma hæmatobium*; ¹ sometimes also, according to Griesinger, it causes severe intestinal lesions, and in certain not at all rare cases (especially in Egypt where the disease is on the whole more severe than at the *Cape*²) it leads to general cachexia and the death of the patient.

Opinions are still divided as to the mode in which the parasite *invades the human body*. The invariable association of the disease with sea-coasts and the shores of river estuaries, contrasting with its absence from the adjoining inland districts where the disease is never seen except in imported cases, renders it highly probable that the eggs or embryos of the parasite either come with the water itself or that they cling to certain aquatic animals (fishes, crustaceans, gastropods or other molluses) which become the media of conveyance; they would thus be introduced with the drinking water or by means of plants or vegetables that had been in the water, or by these parasite-bearing animals; or they would enter by the skin of persons in the water, or

¹ Pruner, in speaking of the urolithiasis common in Egypt ('*Krankh. des Orients*,' p. 272), had already called attention to the severe lesions of the vesical mucous membrane (which are due to distoma), without knowing their connexion with a parasite, and without adverting to the hæmaturia. On the other hand, the statement of Renoult ('*Journ. gén. de méd.*,' An. xi, vol. xvii, 366) as to the endemic occurrence of blood in the urine among the French troops during the occupation of the country by Napoleon, would seem to refer to this endemic hæmaturia.

² According to Batho (l. c., p. 503) urolithiasis is not found at all frequently except in Port Elizabeth, which is the chief seat of the distoma-disease of that region.

even by the urethra, as has been alleged. Against the latter of these views, maintained by Harley and his informants, there has to be taken into account the striking disparity in the number of cases which may be noticed among persons of different age and sex ; and if the mode of access by the digestive organs is on the whole more likely, still the same variations raise many difficulties in the way of that theory also.

§ 99. D. HÆMATOBIUM MOSTLY AFFECTS MALES AND PRINCIPALLY BOYS.

The various *races and nationalities* would appear to be subject to the disease *cæteris paribus* somewhat uniformly. The opinion of Bilharz that it is chiefly the Egyptians (Copts and Fellahs) who suffer from distoma (the negroes very rarely and the Europeans never), and the assertion of Rubidge (in Harley's paper) that the Caffirs enjoy an absolute immunity from it, have been controverted by Griesinger who has often found the parasites in negroes, by Sonsino who has not unfrequently seen them in Europeans, and by Batho who declares that they are just as common in Europeans as in Zulus. It would appear, also, from the numerous cases at the Cape among coolies from Bombay and Madras, that Hindus and Malays are not exempt.

On the other hand, there is almost complete agreement among observers that the parasite occurs very rarely in the *female sex*, the recorded instances of it being mostly in children or young girls. Griesinger has never seen it in women, and Batho says : "Its subjects are invariably of the male sex." Sonsino is the only observer who has seen a few cases in women, one of them being a Turkish woman who had lived a considerable time in Alexandria.

As regards the *time of life*, it is noteworthy that of 17 cases specially mentioned by Sonsino, 13 were in children and 2 in youths. On the same point Batho says : "In Natal the hæmaturia is very prevalent, large numbers of boys being affected. At the capital, Pietermaritzburg, it would appear as if the majority of the male youth suffer

from it. It commences to show itself almost invariably before puberty, and never attacks persons of middle or old age. The limits of age during which its existence is possible are apparently from five years to thirty. I was unable to hear of a single instance in which it commenced at a later age." And a similar opinion is expressed by the authorities for places in Cape Colony (Uitenhage and Port Elizabeth).

§ 100. D. HÆMATOBIUM COMMONEST IN SUMMER.

With respect to the influence of the *season of the year* upon the amount of the sickness, Griesinger says that he found the parasites in one-half of the bodies that were opened in the months of June, July, and August, but in only one fourth of those opened from September to January. He thinks that this is not accidental but connected somehow with the food; it is possible, however, that the fact may be explained by variations in the frequency of the parasites themselves in the several seasons. Without doubt the duration and severity of the sickness depends on the introduction of the worm in considerable quantities and over considerable periods; and, accordingly, the most certain means of cure is to remove the infected person away from the habitat of the parasite.

III. *Nematodes.*

§ 101. ASCARIS LUMBRICOIDES.

In regard to their diffusion over the whole habitable globe and the frequency of their occurrence, *Ascaris lumbricoides*, *Oxyuris vermicularis* and *Trichocephalus dispar* are a group that hold unquestionably the first place among parasitic worms. The round-worm is a parasite that is indigenous wherever men congregate. While statistics do not enable us to give its relative frequency in various parts of the world with mathematical accuracy; yet we may infer from the statements of authorities in *tropical and subtropical regions*,

and from the language of astonishment that they use in speaking of its enormous prevalence, that it is much more common with them than in higher latitudes.

This is the opinion held by Pruner,¹ Hartmann,² Vauvray,³ and others for the *Nile countries*, by Harris⁴ and Courbon⁵ for *Abyssinia*, by Borchgrevink⁶ for *Madagascar*, by Grenet⁷ for *Mayotte*, by Allan⁸ for the *Seychelles*, and for the *Mauritius* by Dyer,⁹ who says: "This complaint is nearly universal in the Mauritius . . . In the black population in such numbers are the lumbrics produced, that I have frequently been disgusted by seeing them crawling from the anus and mouth at the same time. One black literally brought me his hat full, which he assured me he had passed very shortly before." Further, by Daniell¹⁰ for the *West Coast of Africa*, Chassaniol¹¹ for *Senegambia*, Pruner, Robertson¹² and Guys¹³ for *Syria*, Pruner for *Arabia*, Ward and Grant,¹⁴ Voigt,¹⁵ Waring,¹⁶ Day,¹⁷ Huillier,¹⁸ Auboeuf,¹⁹ and others for *India*, Waitz,²⁰ Heymann,²¹ and v. Leent²² for the *East Indies*, Bernard²³ and Beaufile²⁴ for *Cochin China*, Wilson²⁵ and Smart²⁶ for *China*, Friedel²⁷ and Wernich²⁸ for *Japan*, Bernoulli²⁹ for *Central America*,

¹ 'Krankh. des Orients,' 244.

² 'Naturgesch.-med. Skizzen der Nilländer,' Berl., 1866.

³ 'Arch. de méd. nav.,' 1873, Sept., 161.

⁴ 'The Highlands of Æthiopia,' Lond., 1844, ii, 407.

⁵ 'Observ. topogr. et méd., &c.,' Par., 1861, 35.

⁶ 'Norsk Mag. for Laegevidensk.,' 1872, viii, 240.

⁷ 'Souvenirs méd. . . à Mayotte,' Montp., 1866.

⁸ 'Edinb. Monthl. Journ.,' 1841, Aug., 569.

⁹ 'Lond. Med. Gaz.,' 1834, March, 866.

¹⁰ 'Sketch of the Med. Topogr. of the Gulf of Guinea,' Lond., 1849, 53.

¹¹ 'Arch. de méd. nav.,' 1865, Mai, 511.

¹² 'Edinb. Med. and Surg. Journ.,' 1843, April, 247.

¹³ 'Statist. du Paschalik d'Alep,' Marseille, 1853, 63.

¹⁴ 'Official Papers, &c.,' Pinang, 1830.

¹⁵ 'Bibl. for Laeger,' 1834, i, 352.

¹⁶ 'Ind. Annals of Med. Sc.,' 1859, July, 371.

¹⁷ 'Madras Quart. Journ. of Med. Sc.,' 1862, Jan., 37.

¹⁸ 'Arch. de méd. nav.,' 1868, Févr., 87.

¹⁹ 'Contribut. à l'étude . . . des malad. dans l'Inde,' Par., 1882, 70.

²⁰ 'On Diseases incident to Children in Hot Climates,' Bonn, 1843, 263.

²¹ 'Würzb. phys. med. Verhandl.,' v, 30.

²² 'Arch. de méd. nav.,' 1867, Sept., 170.

²³ 'De l'influence du climat de la Cochinchine,' Montp., 1867.

²⁴ 'Arch. de méd. nav.,' 1882, Avril, 265.

²⁵ 'Med. Notes on China,' Lond., 1846, 193.

²⁶ 'Transact. of the Epidemiol. Soc.,' 1862, i, 219.

²⁷ 'Beitr. zur Kenntniss des Klimas und der Krankh. Ost-Asiens,' Berl., 1863, 33.

²⁸ 'Deutsche med. Wochenschr.,' 1878.

²⁹ 'Schweiz. med. Zeitschr.,' 1862, iii, 100.

Levacher,¹ Dazille² and Ruz³ for the *West Indies*, Rodschied⁴ and Bajon for *Guiana*, and by Jobim⁵ and Sigaud⁶ for *Brazil*.

From the highest latitudes also, such as those of *Newfoundland*⁷ and *Greenland*⁸ we have information of the endemic occurrence of *Ascaris lumbricoides*. The single exception to the general rule, so far as I know, is *Iceland*, where this parasite, according to Finsen,⁹ is rarely met with.

§ 102. MODE OF ACCESS OF *A. LUMBRICOIDES* TO THE HUMAN BODY.

There is still some difference of opinion as to the mode of access of *Ascaris lumbricoides* to the human body. Either the embryo-containing eggs discharged from the human intestine may reach the organism in the food or drink, particularly by means of uncooked field or garden produce, thereafter developing to worms,—an alternative which is opposed, no doubt, by the fact that all experiments hitherto to infect men by the introduction of the eggs of *ascaris* have failed;¹⁰ or, as Leukart considers more probable, the transmission takes place through an intermediate host belonging to one of the lower classes of animals. “Considering the frequency and almost universality of the human round-worm,” he says,¹¹ “we may at any rate conjecture that the transmitting agent or agents of it are very generally diffused.”¹²

¹ ‘Guide méd. des Antilles,’ Par., 1840, 193.

² ‘Observ. sur les maladies des nègres,’ Par., 1792, i, 106.

³ ‘Arch. de méd. nav.,’ 1869, Juin, 440.

⁴ ‘Med.-chir. Bemerk. über . . Rio Essequibo,’ Frankf., 1796, 290.

⁵ ‘Disc. sobre as molestias . . do Rio de Janeiro, &c.,’ Rio, 1835.

⁶ ‘Du climat et des malad. du Brésil,’ Par., 1844, 425.

⁷ Gras, ‘Quelques mots sur Miquelon,’ Montp., 1867, 24; and Anderson, in Dobell’s ‘Report,’ 1870, 365.

⁸ Lange, ‘Bemaerkn. om Grønlands sygdomsforhold,’ Kjöbenh., 1864, 43.

⁹ ‘Jagttagelser angaaende sygdomsforholdene i Island,’ Kjöbenh., 1874, 108.

¹⁰ Given by Leuckart (‘Die menschlichen Parasiten, &c.,’ Leipz., 1876, ii, 222).

¹¹ *Ib.*, p. 229.

¹² As these lines were going through the press, I read in a paper by Radu (‘Wien. med. Blätter,’ 1882, No. 45, p. 1386) that he had observed during the general prevalence of *ascaris* in Moldavia in the very rainy year 1881, a case

§ 103. PARASITISM OF ASCARIS PROBABLY FAVOURED BY CLIMATIC HEAT AND MOISTURE.

If it be not definitely proved that *states of soil and weather*, particularly dampness and a high temperature, increase the frequency of the parasites or the prevalence of the malady, yet that becomes highly probable when we consider what is known of the development of ascaris-eggs.¹ The preference of the parasite for negroes, Indians and other uncivilised peoples, is not an affair of *racial or national peculiarity*, but of their manner of life, which is peculiarly favorable to the introduction of the ascaris progeny under the circumstances already mentioned.

§ 104. OXYURIS VERMICULARIS AND TRICHOCEPHALUS DISPAR.

These worms are met with in the same general diffusion over the globe and in the same frequency as *Ascaris lumbricoides*. Many of the above-mentioned observers in tropical or subtropical regions adduce evidence of this in their writings, and there are not less numerous reports establishing the same fact for higher latitudes. There are, no doubt, differences in the prevalence of the worms at various points. Thus Finsen says that in Iceland oxyuris is enormously frequent, contrasting with the very rare occurrence of ascaris; while, contrariwise, Ruzs dwells upon the rarity of oxyuris in Martinique, as compared with the very wide diffusion of ascaris. Virchow² observes that he used to find

which went to prove that the reproduction of the round-worms in the human intestine takes place not merely through eggs reaching it, but also by the fully-developed young brood being liberated alive from the mother's body. From the patient there was discharged a solid mass of gelatinous and opalescent substance, about the size of a hen's egg (a so-called round-worm's nest), adhering to the worm. On examination, it was found that the head of a young ascaris projected from the middle of the body of this worm; and when the young one was pulled out a second followed clinging to it, and then a third, and so on to the seventh. With the last there came away a tube about an inch and a half long; the whole brood was enclosed in a thin and almost transparent sheath.

¹ Leuckart, p. 211.

² 'Archiv für pathol. Anat.,' 1857, xi, 81.

trichocephalus in the course of *post-mortem* examination more commonly at Würzburg than at Berlin. According to Krabbe,¹ the whip-worm is very rare in Copenhagen. But all such data are so isolated, or have been arrived at under such special circumstances, that they have no value for a general conclusion as to the number of cases in various parts of the world.

According to the inquiries of Leuckart,² there can be no doubt as to the introduction of both these parasites by embryo-containing eggs discharged from the human intestine, and without the intervention of any transmitting medium.

§ 105. TRICHINA SPIRALIS: ITS DISCOVERY.

The first trustworthy accounts of the trichina-disease in man reach no farther back than about the year 1830; and even as late as 1860 the observations concerning it related to muscle-encapsulated trichinæ accidentally found in the course of anatomical examination.

Apart from a somewhat doubtful case of Tiedemann's³ in which large calcareous concretions were found in the muscles of a subject, and not reckoning from the muscle-preparations with calcified trichinæ which Peacock⁴ in 1828 deposited in the museum of Guy's Hospital, the first case of encapsuled trichinæ is that recorded by Hilton,⁵ who spoke of them as "oval bodies, transparent in the middle and opaque at their end, altogether about $\frac{1}{25}$ th of an inch in length." These he found in a dissected subject in all the muscles of respiration; "no organisation," he added, "could be discovered with the aid of a microscope;" and his explanation of them was that they were very minute cysticerci. The merit of recognising the parasitic nature of these objects and of accurately describing the parasites themselves belongs to Owen⁶ and Harrison.⁷

¹ Quoted by Leuckart, p. 466.

² L. c., pp. 332, 498.

³ 'Froriep's Notizen,' 1821, i, 64.

⁴ According to Cobbold, 'Entozoa,' Suppl. 1.

⁵ 'Med. Gaz.,' 1833, Feb., p. 605.

⁶ 'Lond. and Edin. Philos. Mag.,' 1835, p. 452, and 'Med. Gaz.,' 1835, April, p. 125.

⁷ 'Dubl. Journ. of Med. Sc.,' 1835, Sept., p. 185.

Owen's discovery was made in a piece of muscle beset with encapsuled trichinæ, which was brought to him by Paget, then a student at St. Bartholomew's Hospital, where the appearance had several times been pointed out to him by Wormald, the prosector. Paget's conjecture was that the objects in question were entozoa. An examination with the microscope bore out this guess; Owen succeeded in making out the parasite, its position within the capsule, and its characters; and it was he that gave it the name of "*Trichina spiralis*."

Harrison gave a demonstration of the parasite in August, 1833, in the medical section of the British Association, then in session at Dublin; and thereafter followed accounts of it by Farre,¹ of London, Knox,² of Edinburgh, Curling,³ Bischoff⁴ (Heidelberg), Bowditch and Wyman⁵ (Boston), Svitzer⁶ (Copenhagen), Bristowe and Rainey⁷ (London), and by Turner,⁸ of Edinburgh, who stated that he had found the parasites in from 1 to 2 per cent. of all the subjects in the dissecting-room during the five years preceding (1855—1860). Meanwhile Leidy⁹ had proved the occurrence of the trichina in the pig, and Guret¹⁰ had found it in a cat; and these discoveries gave rise to the feeding experiments of Herbst,¹¹ Leuckart,¹² and Virchow,¹³ and finally to the distinction made by Virchow,¹⁴ and simultaneously by Zenker,¹⁵ between muscle-trichinæ and intestinal trichinæ, and to a correct understanding of the genetic relation between the two.

The significance of the parasite as a cause of disease in

¹ 'Lond. Med. Gaz.,' 1835, Dec., 382.

² 'Edinb. Med. and Surg. Journ.,' 1836, July, 89, and 'Lond. Med. Gaz.,' 1843, Sept., 805.

³ *Ib.*, 1836, Feb., 768.

⁴ 'Med. Annalen,' 1840, vi, 232.

⁵ 'Boston Med. and Surg. Journ.,' 1841-43.

⁶ 'Froriep's Notizen,' 1847, Nr. 35, 195.

⁷ 'Transact. of the Pathol. Soc.,' 1854, v, 277.

⁸ 'Edinb. Med. Journ.,' 1860, Sept. 209.

⁹ 'Proceed. of the Acad. of Nat. Sc. of Philadelphia,' 1846, Oct., 107, and 'Annals and Mag. of Nat. Hist.,' 1847, xix, 358.

¹⁰ Appendix to part i of his 'Lehrbuch der pathol. Anat. der Hausthiere,' Berl., 1849, 144.

¹¹ 'Gött. gelehrt. Anz.,' 1851, Nr. 19; 1852, Nr. 12.

¹² 'Arch. für Naturgesch.,' 1857, ii, 188, and 'Compt. rend.,' 1859, xlix, 452.

¹³ 'Deutsche Klinik,' 1859, 430, and 'Compt. rend.,' 1859, xlix, 660.

¹⁴ *Ib.*, 1860, li, 13, and 'Arch. für pathol. Anat.,' 1860, Bd. 18, 330, 535.

¹⁵ *Ib.*, 561.

the human body remained quite obscure until 1860. No doubt the connexion between the acute muscular pains felt by the patient, and the presence of parasites found after death had been correctly indicated by Wood¹ in 1835.

Wood's case was that of a young man, æt. 22, who was admitted in October, 1834, into the Bristol Infirmary for "acute rheumatism," implicating the trunk and the extremities, and who died a few days after. The *post-mortem* examination showed commencing pneumonia and pericarditis, and the presence in the muscles, especially in the pectoral and deltoid, and in the muscles of the chest and shoulders generally, of what proved to be the entozoa that Owen had described. Wood was unable to make out the nature of these at the time, although he took the microscope to them; but when he heard of Owen's discovery they became at once clear to him; and, he adds, "would it not be well to ascertain, if possible, whether in either or all of the cases alluded to in Mr. O.'s paper, there was any symptom of rheumatism or inflammation of any kind in the muscular system?"

This idea of Wood's, however, remained unnoticed. The trichinæ, which were afterwards detected so often, were regarded as harmless intruders in the human body, and by some (*e.g.* Bischoff) as products of spontaneous generation; until at length Zenker came forward in the beginning of 1860 with his pioneering observation of a severe case of trichinosis, whereby he proved the connexion between that disease and the eating of trichinous pork, and thus constituted himself, along with Leuckart and Virchow, the founder of the doctrine of trichina-disease.

§ 106. EARLIER CASES OF TRICHINOSIS. PRESENT DISTRIBUTION.

No sooner did the fact become known to the profession than numerous accounts began to come in from North Germany of sporadic cases of sickness due to trichinosis as well as of trichinous epidemics. At the same time evidence more or less trustworthy was adduced that the disease had occurred before, although the nature of it had not been recognised.

Whether the cases of sickness observed by Fehr in 1675, in a peasant's family in Würtemberg, are to be taken for trichina-disease, as some

¹ 'Medical Gazette,' 1835, May, p. 190.

have believed, appears to me very doubtful.¹ Still less can I accept the view of Le Roy de Méricourt, according to which the disease observed in France, in 1828-29, and designated with the name of "acrodynia," was trichinosis.² The instance given by Klopsch³ of a group of trichinosis cases in 1842 is to be accepted, inasmuch as in 1866 living trichinæ could still be detected in the intercostal muscles of one of the persons originally attacked by the disease. Probably also the epidemic of 1849 at Wegeleben, described by Mosler,⁴ and taken to be "English sweating sickness," was an affair of trichinosis; and the same holds for the cases observed at Schwetz in 1858 and reported upon by Meschede.⁵ The mysterious sickening of a whole company who had dined together at a small town in Hesse in 1845, was fortunately explained⁶ (and the suspicion of having poisoned them removed from their host), by the discovery in 1864 of encapsuled trichinæ in the muscles of one of the members of the company, in an operation upon him by Langenbeck. In the same way Tünger⁷ succeeded in proving by a *post-mortem* examination in 1863 on the body of an individual who had been seized at Hamburg in 1851 with the same kind of symptoms as several others in his company, that the illness had been a small epidemic of trichinosis. Griepenkerl⁸ also showed by subsequent anatomical examination that the disease which prevailed at Blankenburg, in the Harz, in 1858-60 among a battalion of Brunswick Jägers, and was set down as "gastric rheumatic fever," was no other than trichinosis. The following⁹ has been communicated to me by Surgeon-General Dr. Abel, of the German army: In 1851 at Halberstadt, Quedlinburg, and the village of Harsleben, situated between those towns, he treated about 150 cases of a remarkable malady which appeared to him at the time to be very mysterious, although when he afterwards came to know the trichina disease he recognised its character. All the cases got well, and he considered the disease to be a peculiar kind of influenza, although he was constrained (according to the symptoms) to locate its seat in the subcutaneous connective tissue and in the fasciæ. All the doctors who practised in Halberstadt, Quedlinburg, and neighbourhood, assured him, in answer to his questions, that precisely the same disease had been known in that locality for several years, that it had been, moreover, often fatal, and had been taken to be a kind of nervous fever. The town of Wegeleben was indicated as the place where the disease had

¹ 'Miscell. med. phys. acad. nat. cur. Leopold,' 1677, Decas i, Ann. vi, obs. 191, p. 269.

² See p. 252, note.

³ 'Virchow's Arch.,' 1866, Bd. 35, 609.

⁴ Ib., 1865, Bd. 33, 414.

⁵ Ib., 1864, Bd. 30, 471.

⁶ Accord. to Lücke, 'Viertelj. für gerichtl. Med.,' 1864, Jan., 102.

⁷ 'Virchow's Arch.,' 1863, Bd. 28, 391.

⁸ 'Deutsche Klinik,' 1864, No. 17. See also Scholz, ib., No. 40.

⁹ [Introduced into the English edition from the author's MS.]

been seen especially often and in its most fatal form. There had been no *post-mortem* examinations. It can hardly be doubted that all these cases were cases of trichina-disease; and the matter is all the more interesting from the fact that the sickness happened in the very district in which the first authenticated epidemics of trichinosis afterwards occurred. The assumption that the outbreak of trichinosis had been caused by the introduction of Chinese swine is quite untenable for the locality above referred to. Besides the indigenous breed, the pigs there for a period of thirty to fifty years have all been importations—in enormous droves—of the Polish breed.

The publication of Zenker's case was followed by notices of cases, either sporadic or in groups, by Waldeck¹ (Korbach in Waldeck), Wunderlich² (Leipzig), Friedreich³ (Würzburg), Sandler⁴ (Magdeburg), Landois⁵ (Rügen), and others. Then came accounts of extensive outbreaks of trichinosis as an epidemic in the following places: Calbe-on-the-Saal⁶ in 1862, Plauen⁷ in 1861-63, Hettstädt⁸ in 1863-64, the Saal circle⁹ in 1864 and following years, Quedlinburg¹⁰ in 1864, Hadersleben¹¹ (one of the severest epidemics), Zoppot,¹² Lübeck,¹³ Konitz¹⁴ and Chemnitz,¹⁵ Neudorf¹⁶ (near Breslau) and Weimar,¹⁷—all in 1865, Berlin¹⁸ in 1867, Schönebeck in 1868, Erlangen¹⁹ and Heidelberg²⁰ in 1870, Bovenden²¹ (near

¹ 'Jahresb. der Gesellsch. für Natur- und Heilkde. in Dresden,' 1861-62, 50.

² 'Arch. der Heilkde.,' 1861, ii, 269.

³ 'Virchow's Arch.,' 1862, Bd. 25, 399.

⁴ 'Deutsche Klin.,' 1862, 261.

⁵ *Ib.*, 1863, Nr. 4, 8.

⁶ Simon, 'Preuss. med. Vereins-Ztg.,' 1864, Nr. 38, 39.

⁷ Böhler and Königsdörffer, 'Die Erkenntniss der Trichinenkrankh.,' Plauen, 1864, and Sequel, *ib.*, 1865.

⁸ Rupprecht, 'Die Trichinenkrankh., &c.,' Hettstädt, 1864.

⁹ Puder, 'Die Trichinen in Halle und im Saalkreise,' Halle, 1870.

¹⁰ Wolff, 'Deutsche Klin.,' 1864, Nr. 16, 18.

¹¹ Kratz, 'Die Trichinenkrankh. in Hadersleben,' Leipzig, 1866.

¹² Benzler, 'Berl. klin. Wochenschr.,' 1865, 51.

¹³ Eschenburg, 'Hannov. Ztg. für pr. Heilkde.,' 1865, 496.

¹⁴ Wolff.

¹⁵ Günther and Flintzer, 'Zeitschr. für Med., Chir. und Geburtsh.,' 1867, 526.

¹⁶ Lebert, 'Gaz. méd. de Paris,' 1866, 195 ff.

¹⁷ Frommann, in 'Virchow's Arch.,' 1871, Bd. 53, 501.

¹⁸ Account in 'Berl. klin. Wochenschr.,' 1867, 357, and Hoffmann, *ib.*, 547.

¹⁹ Maurer, 'Arch. für klin. Med.,' 1871, viii, 368.

²⁰ Friedreich, *ib.*, 1872, ix, 459.

²¹ Kraemer, 'Deutsche Klin.,' 1872, 277, 289, and Seebohm, 'Die Trichinen-Epidemie zu Bovenden,' Dissert., Gött., 1872.

Göttingen) and Löbau¹ (in Saxony) in 1872, Westphalia² in 1877, Hof, Nürnberg, Bamberg, Markt-leuten³ and Crailsheim⁴—all in 1878, and Barmen⁵ in 1880.

By far the larger part of all these notices, and numerous other accounts of isolated cases of trichinosis, relate to *Northern Germany*. The few instances as yet reported from South Germany are, as we have seen, those from Hesse in 1845, Heidelberg and neighbourhood in 1866-70, Erlangen in 1870, and the Franconian towns above mentioned, together with Crailsheim, in 1878. Doubtless there have been many cases of trichinosis both in North and South Germany, which have not come to the knowledge of practitioners or have been wrongly diagnosed, or have been passed over without mention, particularly of recent years when the interest in the matter has declined and the disease itself has become decidedly less common. But we may still safely assume that trichinosis, particularly in its epidemic form, has occurred much more rarely in South Germany than in North; and we may conclude in like manner, from the extremely scanty accounts of the malady that reach us from *Austria*—small epidemic at Brünn⁶ in 1866, sporadic cases at Prague⁷ in 1866, and at Vienna⁸ in 1867—that trichinosis has been seen but seldom in that country. From *Great Britain*—where, as we have seen, the muscle-trichinæ were first discovered in the dead body and repeatedly described in subsequent years—there come notices of only two small epidemics of trichinosis, the one in 1871 in the village of Workington⁹ (Cumberland), and the other in 1879 among the boys on board a training-ship in the Thames.¹⁰

In *Denmark* trichinæ were seen for the first time in the

¹ Kittel, 'Allg. Wien. med. Ztg.,' 1871, 254.

² Müller, 'Deutsche Zeitschr. für pract. Med.,' 1876, Nr. 14, 15.

³ Bollinger, 'Zeitschr. für Thiermedizin,' 1879, v, 13.

⁴ Haeberlein, 'Württbg. med. Correspondenzbl.,' 1879, Nr. 26, 27.

⁵ Strauss, 'Deutsche med. Wochenschr.,' 1880, Nr. 48.

⁶ Boner, 'Allgem. Wien. med. Ztg.,' 1866, Nr. 6.

⁷ Knoll, 'Prager Vierteljahrschr. für Heilkde.,' 1866, iii, 144.

⁸ Peyritsch, 'Wochenbl. der Gesellsch. der Wiener Aerzte,' 1867, Nr. 34; Flamm, 'Wien. med. Wochenschr.,' 1867, Nr. 74 ff., 1868, 139, 730.

⁹ Dickinson, 'Brit. Med. Journ.,' 1871, April, 446.

¹⁰ Power, 'Report of Med. Officer of the Local Government Board,' Suppl. for the year 1879, 47.

winter of 1866-67 in three subjects in the dissecting-room at Copenhagen, and a case was shortly afterwards diagnosed in that city during life.¹ To the same period belong notices of sporadic cases in Denmark,² and two years later there was a case of trichinosis under observation in Fühnen.³ In Sweden, down to 1873, cases of trichinosis had never been seen in groups, but only quite casually; although every year trichinous swine were met with, and trichinæ frequently found after death in the bodies of persons who had died of other diseases.⁴ The first accounts⁵ of trichinos in *Russia* date from 1866; but there was no considerable prevalence of the disease until 1873 at St. Petersburg, 1874 at Moscow and Lodz (Poland), 1878-79 at Riga, and 1879 again at Moscow⁶ and St. Petersburg.⁷ For *Roumania* we have Scheiber's⁸ statement that encapsuled trichinæ had been found after death in a subject at Bukharest; he adds, that trichinosis would appear to be very rare in that country. In *Switzerland*, according to the account by Roth,⁹ trichinæ were first discovered in two cadavers in 1860 by Miescher, and two more cases were found among 1914 corpses examined anatomically in that town during the period from 1872 to 1880. A small epidemic of trichinosis occurred in Switzerland in 1868, at the village of Ravecchia within a mile of Bellinzona in the Canton Ticino.¹⁰ Concerning the occurrence of the malady in *France*,¹¹ *Italy*, *Spain*, and *Portugal*,¹² I know of only occasional notices, and these relate

¹ Krabbe, 'Tidsskrift for Veterinar,' 1867, xv, H. 3.

² Ring, 'Ugeskrift for Laeger,' 1868, v, Nr. 11; Ditlevsen, ib., Nr. 12.

³ Petersen, 'Hospitallstidende,' 1872, xv, Nr. 5.

⁴ Axel Key, 'Hygiea,' 1868, xxx, 127, and 'Sundhets-Colleg. Berättelse åren 1865—1873.' I find no further mention of the disease in the medical reports after this.

⁵ Maydell, 'Petersb. med. Zeitschr.,' 1866, x, 81; Erichsen, ib., 161; Rudnew, in 'Virchow's Arch.,' 1866, Bd. 35, 600.

⁶ Knoch, ib., 1876, Bd. 66, 393, and 'Petersb. med. Wochenschr.,' 1880, Nr. 16.

⁷ Kernig, ib., 1880, Nr. 1.

⁸ In 'Virchow's Arch.,' 1872, Bd. 55, 462.

⁹ 'Correspondenzbl. für Schweizer Aerzte,' 1880, x, 129.

¹⁰ Zangger, 'Wochenschr. für Thierheilkde.,' 1869, xiii, 55, and Jauch, 'Annali univ. di med.,' 1869, Oct., 72.

¹¹ Cruveilhier, 'Anat. pathol.,' Par., 1842, ii, 64; Delpech, 'Bull. de l'Acad. de méd.,' 1866, xxxi, 659.

¹² Silva Amado, 'Bayr. ärztl. Intelligenzbl.,' 1868, 506.

almost exclusively to the detection of the parasite in the pig or in the human body after death. For Spain, the statement is made, *à propos* of the discovery of trichinæ in the indigenous breed of pigs in Barcelona, that epidemics of trichinosis had often been observed before in various parts of the country.¹

North America would appear to be subject to particularly severe visitations of trichinosis, as might have been almost inferred from the enormously frequent occurrence of the parasite in the American hog. Not to mention the evidence of this furnished by the pork imported into Europe from the United States, we find the fact asserted by American practitioners themselves; as, for instance, by Sutton,² who says that in the Western States at least 4 per cent. of all the swine are trichinous. Accounts of the trichina-disease among the people on a large scale reach us from New York³ for the year 1864, from Marion (Iowa),⁴ Chicago⁵ and Springfield⁶ 1866, New York⁷ 1868, Philadelphia⁸ 1869, a locality in Western Virginia⁹ in 1870, Michigan¹⁰ in 1875, and other places.

In the medical literature relating to *Central* and *South America*,¹¹ as well as the *West Indies*, there is, so far as I know, not a word said about trichinosis. Also for the countries of *Asia* and *Africa* I have found but very few references to the disease. One of these is by Wortabet,¹² who gives an account of a severe epidemic of trichinosis at the village of El Khiam in the highlands of *Syria*, not far from the sources of the Jordan, which had been caused by

¹ Quoted by Bollinger in Virchow-Hirsch's 'Jahresber.' 1879, i, 625.

² 'Transact. of the Indiana State Med. Soc.,' 1875.

³ Jackson, 'Amer. Journ. of Med. Sc.,' 1867, Jan., 101.

⁴ Ristine, 'Med. News,' 1866, 111.

⁵ Smith, 'Chicago Med. Journ.,' 1866, 161.

⁶ Report in 'Lancet,' 1867, April, 501.

⁷ Buck, 'New York Med. Record,' 1869, March 7.

⁸ Stockton Hough, 'Amer. Journ. of Med. Sc.,' 1869, April, 565; 1870, Jan., 282.

⁹ Wiesel, 'Transact. of the West Virginia State Med. Soc.,' 1871, 63.

¹⁰ 'Report of the State Board of Health of the State of Michigan, 1876.'

¹¹ Tüngel (in 'Virchow's Archiv,' 1863, xxvii, 421) mentions a case of trichinosis on board a Hamburg vessel, which had been caused by eating of the flesh of a pig shipped at Valparaiso.

¹² 'Lancet,' 1881, March, 454, and 'Virchow's Arch.,' 1881, Bd. 83, 553.

eating the flesh of a wild boar ; in this case the diagnosis was confirmed by Virchow¹ from specimens of the muscles sent to him. As regards the occurrence of trichinosis in *India*, I find only a brief statement that cases of it had been seen in Calcutta. In *China*, according to an unauthenticated account, those of the inhabitants who do not live in cleanliness suffer from trichinosis ;² but there is no corroboration of this fact on the part of medical observers. In *Algiers*, encapsuled trichinæ were found in a dead body in 1867,³ but there is no further information about the malady there ; and all reference to it is likewise completely wanting for every other part of the continent of *Africa*.

§ 107. LIFE-HISTORY OF THE TRICHINA PARASITE. ANOMALOUS DISTRIBUTION OF TRICHINOSIS.

Our acquaintance with trichinosis extends, as we have seen, no farther back than the year 1830. It is only within the last twenty or thirty years that we have obtained a more intimate knowledge of it ; although that applies to only a comparatively small part of the globe. It is all the more necessary to be cautious in drawing conclusions about the origin and geographical distribution of the disease, or about the native habitat of the parasite and its mode of diffusion over the globe, inasmuch as even at the present day, despite the utmost precision of research, the question as to the proper and original *host of the parasite* has not been solved with absolute certainty. There are two animals which enter into competition for this privilege, the pig and the rat ; these are by far the most frequent carriers of the parasite, and it is around them that speculation as to the origin of the disease circles.

If we bear in mind that the first authentic cases of trichinosis in man were observed about the year 1830, and that

¹ *Ib.*, 554.

² 'Lancet,' 1864, Dec., 24.

³ Berkhan, in 'Virchow's Arch.,' 1866, Bd. 35, 9.

⁴ Gaillard, 'Bull. de la soc. de méd. d'Algér., 1867, vi ; also 'Gaz. hebd. de méd.,' 1867, 654.

this incident coincided with, or followed close upon, the importation of the *small Chinese breed of swine* into England and shortly after into North Germany—if we bear this in mind, admitting at the same time that the trichina-disease of man is caused solely by eating of the flesh of trichinous pigs, we shall see much that is probable in the opinion held by Gerlach,¹ Rupprecht² and others, that the parasite was imported with these animals into Europe, and perhaps into the United States also. In support of this theory, Berkhan calls special attention to the fact that some twenty years ago, or about the time when trichinæ became known in North Germany, Chinese swine were introduced for breeding purposes into an estate called Schlaustedt, and into Hornburg on the Brunswick frontier. This theory sets out by assuming what we have no proof of, that the parasite occurs in China at all.

From another side, on the assumption, namely, that the rat is the proper carrier of the trichina, the pig being subsequently infected from it, it has been conjectured that the appearance of the disease in Europe has to be connected with the *invasion of the brown rat* (*Mus decumanus*) from Asia. But, not to mention that the “rat theory,” as pointed out by Zenker,³ rests upon error in any case, it has to be admitted that the brown rat was met with as early as the middle of the last century, whereas cases of trichinosis did not occur until long after. It is a remarkable fact in the history of the trichina-disease, and one that has not been explained hitherto, that no part of the world, excepting perhaps Russia and the United States, has been so severely visited as North Germany. No doubt the accounts from other countries are too defective to enable us to conclude with certainty; but this much holds good, that North Germany has suffered from trichinosis much more than South Germany, and that the Scandinavian countries, which can be shown to have no lack of trichinous swine, have been but slightly affected by the trichina-disease. It is obvious that the reason of these variations in the amount of the disease is not to be sought for in any differences in the ways

¹ ‘Die Trichinen,’ Hannov, 1866, p. 74.

³ L. c., p. 88.

² ‘Arch. für klin. Med.,’ 1871, viii, p. 395.

of living among the nationalities in question. Whether this and other puzzling problems in the history of trichinosis will ever find a complete solution is doubtful, inasmuch as it is hardly possible to answer the cardinal question whether the first occurrence of the disease in Europe and North America does in fact belong to quite recent times. Meanwhile it would be a valued service to scientific research to make out the extent to which trichinosis, both of men and animals, is met with in other countries besides Europe and the United States of America.

Anchylostoma duodenale and the Anæmia associated with it.

§ 108. SYMPTOMS OF THE CACHEXIE AQUEUSE OF WEST INDIAN NEGROES.

At the beginning of the eighteenth century the attention of French and English observers was drawn to a very destructive form of disease prevalent among the negroes of the West Indies and Guiana. One set of symptoms pointed to disorder of the intestinal canal,—oppression or pain in the region of the stomach, want of appetite alternating with ravenous hunger, pica (or depraved taste for things inedible and particularly for earthy substances), obstinate constipation, and at a subsequent stage diarrhœa. Another series of symptoms arose out of the extreme anæmia, namely, palpitation, dyspnœa after slight bodily exertion, small, soft, and irregular pulse quickening upon active movement, coldness of the skin and pallor (appearing in the negro as brownish or greenish discoloration), paleness of the mucous membranes, progressive weakness culminating in complete exhaustion, diminution of the secretions, wasting and dropsy. Either set of symptoms was characteristic, death in many of the cases having been ushered in by colliquative phenomena, hæmorrhages, and dropsical effusions into the meninges or into the lungs. The same malady was afterwards met with, as we shall see in the sequel, at other parts of the globe—in Brazil, Egypt, the West Coast of Africa, and elsewhere—

and not only among negroes, but among people of every race and nationality. According to the prominence of the several symptoms, it has been variously named as follows: mal d'estomac, mal de cœur, dirt-eating, hypohæmia or anæmia intertropicalis, oppilação or conção (Brazilian), cachexia Africana, cachexie aqueuse (French).

§ 109. GEOGRAPHICAL DISTRIBUTION OF CACHEXIE AQUEUSE.

The earliest accounts of this disease are to be found in the narrative of travels by Père Labat¹ of Guadeloupe, and in the history of the British colonies in the West Indies by Bryon Edwards;² the latter was many years a planter in Jamaica, and attributed most of the deaths among the negroes to two diseases,—trismus nascentium and “mal d'estomac.” Then follow numerous scientific reports³ by English, French and Danish practitioners of the last and present centuries who had had opportunities of observing the malady on several of the *West Indian Islands*,⁴ as well as by practitioners in French, English and Dutch *Guiana*.⁵ Of somewhat later date are the notices relating to this form of malignant anæmia from *Brazil*,⁶ where it is prevalent over the whole of the country excepting the most southern or sub-tropical provinces—equally on the coast and in the valleys and elevated regions of the interior. Further, we have accounts of the occurrence of the disease in other parts of the Western Hemisphere—in the valleys of the upper basin of the Marañon in Northern *Peru*,⁷ among the natives of

¹ ‘Nouv. voyage aux isles de l’Amérique,’ Par., 1742, ii, 11.

² ‘Hist. of the Brit. Colon. in the West Indies,’ Lond., 1793 (quoted by Imray).

³ An alphabetical list of the authorities is given at the end of the section.

⁴ Hunter, Mason, Telford, Gregory and Ferguson for Jamaica; Desportes and Chevalier for Domingo; de Cordoba for Porto Rico; Dons for St. Thomas; Grall for St. Martin; Moreau de Jonnés and Duchassaing for Guadeloupe; Savaresy, Noverre, Carpentin and Rufz for Martinique; Imray for Dominica; Levacher for Sta. Lucia; Chisholm for Grenada; McCabe for Trinidad.

⁵ Bajon and Segond for Cayenne; Redschild and Hancock for Brit. Guiana; Cragin, Hille, Landré, v. Leent (l. c., 1880) for Surinam.

⁶ Jobim, Sigaud, Rendu and St. Hilaire, Wucherer, de Rocha, Vauvray, de Moura, Souza-Vaz.

⁷ Castelnau.

Sarayacu on the pampas of the Sacramento in Eastern *Bolivia*,¹ and, if only to a slight extent, among the negroes in some parts of the *Southern States* of the American Union.² In the Old World we meet with one of the more considerable centres of "cachexie aqueuse" on the *West Coast of Africa*, where it is confined to Senegambia³ and the Guinea Coast,⁴ so far as our present information enables us to judge. It is altogether doubtful whether the malady occurs in *Algiers*.⁵ On the other hand it is very widely spread in *Egypt*,⁶ whence we obtain those invaluable accounts of cachexia Africana by Griesinger, which first threw light on the nature of the disease. From the *East Coast of Africa*, also, there are notices of it relating to the Island of *Mayotte*⁷ (Comoro Group) and to the *Zanzibar Coast*.⁸ In Asiatic territory, this specific form of anæmia would appear to be very rare; at all events I have found in the whole of the extensive medico-topographical literature of *India* and the East Indies only a single notice which possibly relates to it, namely, one by Day for Cochin.⁹

¹ Galt.

² Chabert and Duncan for Louisiana; Lyell for Alabama and Georgia. The accounts cited by Heusinger ('Die sogenannte Geophagie, &c.,' Cassel, 1852) of Geddings for South Carolina, as well as the statements of Little ('Amer. Journ. of Med. Sc.,' 1845, July, p. 70) and Letherman (in Coolidge's 'Report on the U. S. Army, &c.,' Washington, 1856, p. 333), both from Florida, relate undoubtedly to the malarial cachexia.

³ Moulin, Thaly, Borius.

⁴ Clarke; also Stormont, who elsewhere confuses the disease with malarial cachexia.

⁵ The account by Catteloup ('Mem. de méd. milit.,' 1852, viii, 1), to which Heusinger makes reference, relates to malarial cachexia. Langg, who speaks also of the prevalence of cachexia Africana among the French troops in Algiers ('Bibl. for Laeger,' 1847, Oct., p. 290), says:—"Sumpfebrène ere derfor endnu stadigt Armeens Svøbe, isaer da det Mandskab, som overstaar dem, efter flere Recidiver naesten aden Undtagelse angribes af den afrikanske Kachexie."

⁶ Sonnini, Savaresy, Hamont et Fischer, Röser, Pruner, Clot-Bey, Griesinger, Isambert.

⁷ Monestier, Grenet.

⁸ Lostalot-Bachoué.

⁹ The notices which Heusinger quotes from Bontius ('Medicina Indorum,' cap. xi) for Java, and from Twining ('Clin. Illustr. of the Diseases of Bengal,' 2nd ed., Calcutta, 1835, i, 399), refer undoubtedly to malarial cachexia. When this sheet was in the press, I found a notice by McConnell ('Lancet,' 1882, July, p. 96), according to which he had seen since 1879 twenty cases of anchylostoma in persons who had died of severe malarial disease, dysentery, pneumonia and

The authorities for the countries of *Nearer and Central Asia* (Syria, Arabia, Persia, &c.), as well as for *China and Japan* are quite silent about it. In the *East Indies*, however, it is met with; being somewhat frequent, as Van Leent¹ states, among the convicts working in the mines in Borneo.²

In Europe, the disease on a considerable scale has been observed hitherto in only one country, namely, *Italy*. The first information about it from that country is in the account by Volpato, who met with it in several rural communes of the district of Treviso (*Venetia*), especially in children; he described it under the name of "allotriofagia" = "mangiare sostanze non alimentari," knowing nothing at the time, of the writings of previous observers.³ Thereafter came accounts of isolated cases at Ponte Buggianese⁴ and Florence⁵ (*Tuscany*), at Turin⁶ (*Piedmont*), and at Cesena⁷ (Province of Forlì in the *Æmilia*). But it is in *Lombardy* that the disease is most widely spread, and indeed truly endemic, particularly in the provinces of Milan and Pavia;⁸ and to that focus of the sickness there belongs the very severe and often described⁹ outbreak of the disease among the labourers employed in making the St. Gothard tunnel, an outbreak which did not spare the workmen even on the northern or Swiss side of the mountain.¹⁰

It is probable that the area of the malady extends farther than we at present know, as Perroncito, Grassi and Parona,

the like. In only eight of these was there anæmia; and the author would assign the cause of that condition, not to the parasitic trouble, but to the diseases above mentioned. He had never seen blood in the stools in these cases.

¹ L. c., 1867.

² Bugnion ('Revue méd. de la Suisse romande,' 1881, Mai, p. 274) mentions a case of the disease observed in 1879 by Roth of Basel in a man who served a long time as a soldier in Java and Borneo, and had returned to Europe invalided in 1878.

³ Heschl gives an account of a case of the same kind, observed at Vienna, in a miner who had been born and had lived in Lower Austria; the disease had been contracted, as Heschl thinks, during a somewhat prolonged stay at Udine, which is also in Venetian territory.

⁴ Morelli.

⁵ Sousino.

⁶ Bozzolo, 'Giorn. internat.,' l. c.

⁷ Cantu.

⁸ Grassi e Parona, Ciniselli. Perroncito ('Revue méd.,' l. c.) also gives an account of a case which he saw at Mantua.

⁹ Concato, Perroncito, Bozzolo e Pagliani, Parona, Pistoni.

¹⁰ Sonderegger, Bäumler, Schönbächler, Immermann (in Bugnion, l. c., 274).

Bozzolo and others are inclined to think. Perroncito¹ has learned from Messieurs Shillinger and Thöt that the same disease has been seen among the miners in Kremnitz and Schemnitz (*Hungary*) ; and the evidence that he adduces of its occurrence among the miners at St. Etienne in *France* (Dept. Loire), has recently been confirmed in a paper by Trossat and Eraud.² It would thus appear that there is good foundation for Perroncito's opinion, based on the observations made hitherto, that many cases of the so-called "miners' disease" fall to be included under this form of anæmia, and that the further experiments and *post-mortem* observations which have been instituted specially with a view to the etiological questions referred to below, may be expected to result in a considerable extension of the area of distribution, and in the annexation of a good many of the cases now classed as "idiopathic anæmia."

§ 110. THE CACHEXIA PROVED TO BE ASSOCIATED WITH ANCHYLOSTOMA DUODENALE.

As to the *cause of the malady*, the views obtaining down to the latest date have been very obscure ; and the obscurity has been all the greater that the disease has been confused with other forms of sickness characterised by the phenomena of general cachexia, more particularly with the malarial cachexia.

This error occurs, as we have seen, in many of the older authors, particularly in Heusinger, who has actually named the disease "malarial chlorosis." More recent observers also, such as Duchassaing, and even some among them, such as Marchand, who are not unacquainted with the true cause of the malady, confound it with malarial cachexia. The most decisive fact is that the morbid conditions of the abdominal viscera (liver and spleen) which are peculiar to the malarial cachexia, have *never* been found, the accounts given even by the earlier observers (Dons, Rendu, Segond, Mason, Pruner and others) bearing witness to this. Another pertinent fact is that the disease has been met with especially often in the negro, who, it is well known, enjoys a decided immunity from malaria.

¹ 'Centralbl. für die med. Wissensch.,' l. c.

² 'Lyon méd.,' 1882, Nr. 25, 217 ff.

At first it was believed that the occurrence of the disease among negroes could be explained by their practice (arising either out of bad habit or of a suicidal purpose) of eating dirt or other indigestible substances.¹ Others laid the stress upon malarial influences, and still others upon insufficient or bad food, chills, bad sanitation, depressed states of feeling (home-sickness), and the like. Griesinger was the first to throw light upon the nature of the “cachexia Africana,” when he showed from his observations in Egypt in the case of fellahs and soldiers, that we had here to do with a *parasitic malady*,—with the presence of *Anchylostoma duodenale* in the intestinal canal of the patient, and with the effects resulting therefrom.

The parasite, as is known, was first described by Dubini² of Milan in 1843, having been found by him *post-mortem* in a considerable number of bodies. Pruner³ afterwards mentioned that he had found it in patients in Egypt; and although he adds that “among adults, it is particularly the cachectic, dropsical, and serofulous who suffer from *Anchylostoma duodenale*,” he did not arrive at a correct view of the relation between the parasites and the “cachexie aqueuse” which he had himself described (l. c., p. 324). Bilharz⁴ discovered the parasite in Egypt anew; and it was in conjunction with Bilharz that Griesinger developed his doctrine of the parasitic nature of cachexia Africana.

This discovery of Griesinger’s was afterwards corroborated by Wucherer, Vauvray, Sonza-Vaz, and others with respect to the Brazilian disease previously known under the name of “hypohæmia intertropicalis,” or “oppilação.” Vauvray remarks that, since attention had been called to the circumstance by Wucherer, the worm had been found in every case of cachexie aqueuse in Brazil which ended fatally. Further confirmation of the fact was supplied by the inquiries of Camuset and Kérangal-Riou in Cayenne, the latter remarking that although he leaves it undecided whether the negro disease known as “mal de cœur” is

¹ This is a transposition of effect and cause. Pica is known to occur often in chlorotic women; and, moreover, it is not an invariable sign of the disease which we are considering. It has been noted also by Marchand as a symptom of disease among the French convicts in Cayenne; and Volpato, as we have seen, has observed it in Treviso.

² ‘Annali univ. di med.,’ 1843, April 5.

³ ‘L. c., 244.

⁴ ‘Zeitschr. für wissenschaftl. Zoologie,’ 1852, iv, 55.

exactly covered by the parasitic malady, yet he could give his assurance that he had not missed finding the parasite in the intestine in a single case which had ended fatally with the symptoms of that kind of anæmia, whether the patient were a negro or of another race. The same opinion has been expressed by Monestier and Grenet for Mayotte, by Borius for Senegambia, by Cantu for Cesena, by the whole of the Italian and Swiss physicians who have observed the disease among the workmen at the St. Gothard tunnel, as well as by Perroncito¹ and by Trossat and Eraud for the miners of St. Etienne. Indeed the most recent observations go to show that the diagnosis does not rest merely upon finding the worm *post-mortem*, but may be also based upon the detection of its eggs in the patient's evacuations.

The parasite hangs on to the intestinal mucous membrane of its host and sucks its nourishment from the blood. Enormous numbers of them, increasing up to several hundreds, take up their abode in the intestine; hence the explanation of the symptoms of irritation in the digestive organs as well as the symptoms of ever-increasing anæmia.

§ III. CIRCUMSTANCES FAVORABLE TO THE INTRODUCTION OF THE PARASITE.

The diffusion of the disease, therefore, is dependent solely on the existence of the parasite and on its introduction into the human organism. According to Leuckart's² showing for *Dochmius trigenocephalus* of the dog, and Perroncito's for the *Anchylostoma duodenale* of man, the mature eggs of the worm, on their discharge from the patient's intestine, undergo their first development in wet soil, being especially favoured by high temperature; and thus the parasite comes in its larval stage, and doubtless by the medium of the drinking-water, into the human intestine, where it completes its development. We thus explain without difficulty how important for the occurrence of cachexic aqueuse are certain *states of soil*, and certain

¹ In the subjects which he examined, Perroncito found, besides *Anchylostoma duodenale*, also *Anguillula stercoralis* and *A. intestinalis*. See the next section.

² L. c., ii, 433.

modes of life, which have often in former times had an erroneous part in the causation assigned to them. Inasmuch as the development of the larva from the egg depends essentially upon dampness of the soil, the prevalence of the disease on *marshy ground* becomes intelligible; although the malady has not on that account anything to do with the malaria which exists or may develop in or upon the ground.

Another fact explained by the natural history of the parasite is that the anchylostoma disease is much commoner *in the country* than *in towns* where the drinking-water would be drawn chiefly from deep wells or large conduits. This applies especially to those dwellers in the country who are occupied in *gardening* or *farming*, as well as to *navvies*, *miners*, and the like, who are oftenest exposed to infection on account of taking their drinking-water from shallow pools and water-courses. Many examples of this are furnished by Wucherer for Brazil, by Bozzolo for Turin, by the Italian and Swiss practitioners from cases occurring among the workmen employed at the St. Gothard tunnel, and by Perroncito and Trossat for St. Etienne.

Bozzolo points out, with reference to the endemic prevalence of the parasite among the workmen at the brickworks in the neighbourhood of Turin, that these men live with their families close to the brickworks; that they get their water supply from springs that collect and form puddles in the loose soil; and further, that they go straight to their meals from digging out the clay or from kneading the bricks, and are accordingly exposed to the risk of introducing the larvæ of the parasite into their mouths by their unwashed hands.¹

The less careful a person is in the matter of cleanliness in general, the more is he exposed to the risk of introducing the parasite; and it is this that explains the fact of the anchylostoma disease being rare among the classes in better circumstances, and confined mostly to the *proletariat* and the *working class*; accordingly, where the population is a

¹ I may direct attention here to a notice by Rühle ('Deutsche med. Wochenschr.,' 1878, No. 46, p. 571) of a form of "pernicious anæmia" which he observed in the neighbourhood of Bonn among persons occupied in brick-making, and to which he accordingly gave the name of "bricker's anæmia." In one of the cases with a fatal issue, the anatomical examination showed nothing remarkable. The question arises whether the intestine had been searched carefully enough.

mixture of nationalities, it prevails especially among those races and nationalities which consist mainly of the latter class. On the other hand we may gather from the facts stated in the foregoing sketch of the disease that *circumstances of race and nationality* do not of themselves occasion either a predisposition to the disease or immunity against it.

A question leading to an interesting train of speculation has been raised by Sonderegger, whether, namely, the occurrence of anchylostoma among the workmen on the Swiss side of the St. Gothard tunnel may not be attributed possibly to *transmission of the parasite* by Italian workmen who had acquired the malady at home. He supposes that the eggs of anchylostoma which are present in the dejecta of Italians suffering from the disease would reach the mud of the water-channels in the tunnel, and that the Swiss workmen and engineers would acquire the infection through their hands and faces getting bespattered with the contents of these channels, and perhaps also through the food getting polluted. The possibility of such a sequence of events cannot well be contested; and we may be permitted to conjecture that the disease can show itself, and attain to general diffusion, in localities where the parasite is not indigenous, but in which it finds the conditions required for its development. All that has been learned hitherto of the history of the anchylostoma disease, does not enable us, unfortunately, to decide whether a diffusion of the disease has already taken place anywhere after that fashion.

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Anguillula stercoralis and the Endemic Diarrhœa of Cochin China.

§ 112. QUESTION OF THE RELATION BETWEEN THE DIARRHŒA AND THE PARASITE.

Various opinions, it is well known, are held by observers as to the etiology of the pernicious chronic intestinal catarrh that occurs in many parts of the tropics. In so far as the question concerns the *diarrhœa of Cochin China*, of specially evil repute, Normand¹ thinks that he has solved it by the detection in the patient's intestine of a parasite belonging to the class of the Anguillulæ, to which he has given the name of *Anguillula stercoralis*. As physician to the naval hospital St. Mandrier at Toulon, he has had many opportunities of observing patients who had acquired that form of diarrhœa in Cochin China and had been invalided home; and he is convinced that this parasite is present in almost every case, and often in enormous numbers.

¹ 'Compt. rend.,' 1876, vol. 83, 316, and 'Arch. de méd. nav.,' 1877, Janv., 35, Févr., 102.

Bavay,¹ who has given an account of the natural history of *Anguillula stercoralis*, found along with it in the patient's intestine, but in smaller quantity, a large and a small variety of another worm which he designates *Anguillula intestinalis* to distinguish them from the other.

According to Normand, the parasite discovered by him occurs only in the Cochin China diarrhœa, being invariably absent in other cases of acute or chronic intestinal catarrh; and this statement Laveran² has been able to confirm in every respect through his investigation of patients returned from Cochin China. Dounon³ also has found the parasite under the same circumstances, associated with still other intestinal worms (*Strongylus*, *Oxyuris*, &c.); and, in his view, there are no fewer than six parasites concerned in producing the Cochin China diarrhœa.

There can be no doubt, after what has been said and remains to be said, of the existence of the entozoon under consideration and of its peculiar characters; but it remains an open question whether the worm is indeed to be considered the true cause of the disease, and not rather a more or less accidental epiphenomenon. The latter view is the one adopted by Liberman,⁴ who declares the anguillula to be not the cause, but the consequence of the intestinal malady, inasmuch as the parasite would find a good feeding ground in the catarrhal secretions of the intestine. Chastang,⁵ who gives a full account of some twenty-two cases of chronic diarrhœa observed by him in Saigon (Cochin China), concludes from his observations that the parasite is found only in exceptional cases even in the native habitat of the disease. His words are:

“Pour ma part (et beaucoup des médecins de Cochinchine avaient la même opinion que moi) je ne crois pas encore à l'origine parasitaire de la diarrhée parce qu'on ne trouve presque jamais (pour ne pas dire jamais) l'Anguillule dans la période d'invasion de la maladie en Cochinchine; et

¹ *Ib.*, 1877, Janv., 36, Juill., 64.

² ‘Gaz. hebd. de méd.,’ 1877, Nr. 42, 116.

³ ‘Traitement de la diarrhée de Cochinchine et de ses affections parasitaires,’ Toulon, 1877.

⁴ ‘Gaz. des hôpit.,’ 1877, p. 237.

⁵ ‘Arch. de méd. nav.,’ 1878, Juill., p. 29.

je crois, ou je serais peut-être mieux porté à penser, que ce parasite n'est qu'une coïncidence ou un résultat des désordres organiques des tuniques intestinales, parce que c'est à la période d'état ou d'aggravation que nous l'avons trouvée quelquefois à Saïgon, et qu'on l'a observée si fréquemment à l'hôpital Saint-Mandrier."

Beaufils¹ also is unable to decide from his experience at Vinh-Long (Cochin China) whether the parasite is the cause or an accidental concomitant of the disease. Mahé² found the anguillula in only a few of the patients admitted into the hospital at Brest with Cochin China diarrhœa; and Chauvin,³ who succeeded Normand in the naval hospital at Toulon, found it only twice in eight cases, while he met with it also in two patients with chronic diarrhœa who had been invalided home from Martinique. A particular interest attaches to the case given by Eyssantier,⁴ of a navy surgeon who was admitted into hospital at Toulon suffering from the diarrhœa of Cochin China; neither Normand nor Bavay had been able to find the parasite in the stools, and it was not until the patient had been three years in France and was convalescent, that the anguillula began to appear in the stools in considerable numbers. Lastly, it is worthy of note that Perroncito⁵ has lately found the *Anguillula stercoralis* and *intestinalis* in several Italian workmen suffering from anchylostoma, who had been employed in making the St. Gothard tunnel.

The importance of Normand's discovery would, therefore, appear to be a good deal open to question. A safe conclusion upon it is not to be looked for until further observations have been made, not only in Cochin China, but also in other tropical countries subject to chronic intestinal catarrh.

¹ Ib., 1882, Avril, p. 264.

² Ib., 1879, Mai, p. 347.

³ Ib., 1878, Févr., p. 154.

⁴ 'L'hôpital maritime de Saint-Mandrier (près de Toulon) pendant l'année 1878,' Paris, 1880, p. 29. The author gives a second case of *Anguillula stercoralis* in a patient who had returned from Martinique with chronic diarrhœa.

⁵ 'Compt. rend.,' 1880, and 'Il Morgagni,' 1880, ll. cc.

Filaria sanguinis hominis.

§ 113. IMPORTANCE OF LEWIS'S DISCOVERY.

One of the most interesting discoveries of recent parasitology, which may be set beside the pioneering work of Griesinger on the distoma disease (or the endemic hæmaturia of Egypt and the Cape due to that parasite), is the detection of a species of filaria in the vascular system of man. This filaria was first discovered in Brazil by Wucherer, in the urine of hæmaturia; it was afterwards detected by Timothy Lewis, in India, in the blood of patients suffering from hæmaturia, and was named by him *Filaria sanguinis hominis*. A long series of investigations have yielded facts of a remarkable kind as to the nature or life-history of the parasite, and as to its relations to other forms of disease, all of them pointing to affection of the lymphatic system. In view of these discoveries one cannot forbear conjecturing that many other diseases of the tropics, which have hitherto remained obscure in their origin, may find their explanation in the detection of this same filaria. The interest attaching to this discovery, therefore, would warrant us in going more minutely into the history of it than the scope of the work demands.

§ 114. HISTORY OF THE RESEARCHES ON ENDEMIC HÆMATURIA AND CHYLURIA.

The first accounts of *endemic hæmaturia and chyluria* date from 1812, in which year Chapotin published a paper on the occurrence of the disease in the Mauritius.¹ His information was afterwards confirmed by Salesse and Rayer, and amplified by accounts of the same endemic malady in Réunion by Quevenne and Mazaé-Azéma. About the same time the

¹ A list of the authors quoted is given in alphabetical order at the end of the section.

attention of practitioners was drawn to the disease in Brazil, where it had long been known to exist; and an animated discussion took place at the Medical Society of Rio on August 15th, 1835, which was, however, pervaded by obscure views as to the nature of the malady.¹ Even in the subsequent notices of hæmaturia and chyluria in Brazil by Sigaud, Juvenot, and Plagge, we find no progress towards an understanding of the disease; and it was not until 1866 that Wucherer furnished an explanation of the nature of this peculiar affection by proving that a parasite occurred in the bloody urine of the patients. Stimulated by Griesinger's discovery of the *Distoma hæmatobium* in the hæmaturia of Egypt, he searched for the ova of that parasite in the urine of patients suffering from hæmaturia at Bahia. He failed to find them; but in their place he found an extremely delicate thread-like worm, belonging to the group of Nematodes. At first he attached no material importance to this discovery; but subsequent observations taught him that the parasite was an invariable factor in the disease, and two years later (1868) he made his discovery public. It found support at once in the clinical history published by Crevaux of a creole patient in Guadeloupe, who was subject to chyluria.

Meanwhile Lewis, before he could possibly know anything of the discovery of Wucherer, had found, at Calcutta in 1868, in the urine of a patient suffering from chyluria "the embryo of a nematoid worm, which may give a clue to one cause of this curious malady."² Two years later he detected the same larval worm in the blood of a patient with chronic diarrhœa,³ and after another interval of two years he found it both in the blood and in the lymphatic fluids of persons suffering from elephantiasis of the legs or scrotum, as well as in the blood of hæmaturic patients.⁴ From these various observations Lewis concluded that it was no mere accidental coincidence which he had come upon, but that an intimate connexion subsisted between the chyluria and the elephantiasis—such as

¹ See the report of it in the 'Revista med. flum.,' l. c.

² 'Annual Report of the Sanitary Commissioner for India,' 1869.

³ 'Report,' 1872.

⁴ 'Report,' 1874, and in 'Indian Annals.'

had already, indeed, been indicated by Simoni, Jobim, and other practitioners in Brazil,¹ by Mazaé-Azéma in Réunion, and particularly by Vandyke Carter in India.³ Lewis's conclusion was *that tropical hæmaturia (not including the distoma disease) and lymphatic elephantiasis were parasitic maladies which depended on the presence of the worm named by him Filaria sanguinis hominis*; and he added by way of conjecture, that they were brought about through the blocking of the smallest blood-vessels and lymphatics by the parasites. These observations were followed by the paper of Sonsino, who found the filaria in Egypt, in the first instance in the urine of a hæmaturic patient (1874) and afterwards in the blood of a person affected with elephantiasis of the scrotum. Next came the discovery of the parasite in the negro skin disease called "craw-craw" (of which more in the sequel) by O'Neill, on the West Coast of Africa, and by Arango³ at Bahia; and lastly Winckel's detection of it in the chylous ascitic fluid drawn from a woman who had lived for ten years in Surinam.

Manson, who was occupied with an inquiry on the occurrence of filaria in Amoy (China), was led by what Lewis taught of the filaria-disease to amplify the view of it somewhat; he showed⁴ that the parasite was found not only in hæmaturia (or chyluria) and elephantiasis but also in other forms of disease dependent on affections of the lymphatic system—in the condition named by him lymph-scrotum, in chylous hydrocele, varicocele, &c.; that it was not so much a matter

¹ See 'Revista med. flum.' It is not improbable that the form of disease described by Bourel-Roncière ('Arch. de méd. nav.,' 1873, Mai, p. 335) under the name of "Lymphangitis primitive de Rio de Janeiro," or of "érysipèle de Rio de Janeiro," is to be included herewith.

² Carter gives a case of chyluria (or hæmaturia) accompanied with varix lymphaticus and elephantiasis, and he explains the connexion between those three affections in this wise, that, if we suppose a certain feebleness in the walls of the lymphatics and blood-vessels, these would become varicose under the pressure of the contained fluid, would rupture in the end, and so give rise to an escape of lymph or blood into the kidneys, ureters, or bladder. We meet with the same view in the writings of later observers, who were acquainted with the filaria and were in a position to associate the lesions of the vessel-walls with that parasite.

³ 'Memoria,' 1875.

⁴ 'China Customs' Gazette,' and 'Med. Times,' 1875, l. c.

of blocking the blood-vessels but rather of stopping up the larger lymphatic vessels, perhaps even the thoracic duct itself in certain circumstances ; that this blocking, however, could not be caused by the larvæ of the parasite (which were the only form of it hitherto seen by any observer) inasmuch as these were so very minute that they could pass through the smallest vessels with ease ; but that it was here a question of the parent animal or the mature parasite, which had hitherto eluded all attempts to find it. Shortly after the publication of Manson's paper, this defect was supplied through the observations made almost simultaneously by Bancroft¹ in Brisbane (Queensland), by Lewis² in Calcutta, by Silva Arango³ in Bahia, and by Dos Santos in Rio. Bancroft found the mature parasite first in a lymphatic abscess on the arm, and in a case of hydrocele ; afterwards⁵ in tumour-like enlargements of the lymphatic glands, in orchitis, lymphangitis, &c. Lewis detected it first in a case of varicose elephantiasis of the scrotum, within a blood-clot that had formed after the swelling had been incised ; along with it he found numerous embryos in the varicose lymphatic vessels and in the fluid of a co-existing hydrocele. Dos Santos found it in a lymph-abscess on the arm ; and Silva Arango met with it in a patient who had suffered from hæmaturia for several years, had afterwards become affected with elephantiasis of the scrotum, and had finally developed the skin disease above referred to under the name of "craw-craw." Quite recently Hillis has found the parent animal in the urine of a hæmato-chyluric patient belonging to Demerara.

§ 115. LIFE-HISTORY OF FILARIA : THE MOSQUITO ITS INTERMEDIATE HOST ; PERIODICITY OF THE PHENOMENA.

These facts having been ascertained (and in part confirmed and amplified by Cobbold, Fayer, and other authorities in Europe, in examining the preparations sent to them from the

¹ L. c., 1877.

² L. c., 1877, p. 78.

³ 'Gaz. da Bahia,' 1877, l. c.

⁴ 'Pathol. Trans.,' 1879, p. 407.

tropics) the question next arose, *how does the parasite find its way into the human body?* Bancroft, in a letter to Cobbold,¹ had, with much acuteness, propounded the question whether the mosquito did not play an important part in these events: "I have wondered," he says, "if mosquitoes could suck up the hæmatozoa and convey them to water. They appear to die in water. I will examine some mosquitoes that have bitten the patient to see if they suck up the filariæ." The question was answered in the affirmative a few months later by Manson,² and placed on a basis of exact investigation. He got a patient affected with filaria to sleep in a room which was lighted up in the evening, the windows being left open; after the mosquitoes had entered in swarms the room was shut up as completely as possible. In the morning he found the walls covered with hundreds of female mosquitoes filled with blood to the bursting (the male mosquito does not live upon blood and has no sting); and the microscopic examination showed that their stomachs contained large numbers of living filaria-embryos, very many more indeed than would be present proportionately in the circulating blood. The following are the conclusions of Manson's inquiry into the subsequent development of the parasite: Many of the larvæ sucked in by the mosquito are digested in its stomach; others undergo a development in the course of a few days to the perfect worm; the mosquito when it is full of eggs resorts to water to deposit them and there perishes, so that the parasites, set free by the death of their host, find their way into the water, and thence into the human body. According to Manson's conjecture they enter through the skins of those who come to the infected water to bathe; having thus found their way into the body they continue their progress until such time as they have reached the blood-vessels or lymphatics. Within the human organism they propagate by way of sexual reproduction; but the development of the larva to the full-sized worm depends upon that sequence of events in which the mosquito co-operates, as above described.

Aranjo,³ and more recently Myers, who repeated in

¹ 'Lancet,' 1878, l. c.

² 'China Customs' Gazette,' 1877, l. c.

³ 'Gazetta da Bahia,' 1879, l. c.

Formosa the experiment with a filaria-patient as devised by Manson, have confirmed the latter's account of the absorption of the larval filariæ by the blood-sucking mosquitoes ; and Cobbold, who has drawn up a detailed description¹ of the metamorphosis of the parasite after Manson's observations, points out² that this developmental process in *Filaria sanguinis hominis* has an analogy in the life-history of *Filaria medinensis*, which has been shown by Fedschenko to go through the stages of its development in the fresh-water cyclops. Magelhães believes that he has found the parasites in the water of a small brook named Carioca, near Rio de Janeiro ; but the statement lacks confirmation. Again, there is no evidence, so far as we have gone, to show whether the parasite enters through the human skin, as Manson assumes and Aranjo and Silva Lima agree, or whether it may not more probably reach the human organism through the drinking of infected water, according to the analogy of *Filaria medinensis*.

There is one very remarkable phenomenon in the life of the parasite, likewise pointed out first by Manson, which enables us to understand how it was that the earlier observers, examining the same patient on different occasions, sometimes found the larvæ in the blood in large numbers and at other times not a single specimen of them. The fact is that in the night-time, or when the patient is asleep, they swarm in the blood, whereas during the day they disappear almost entirely. This observation also has been confirmed in every respect by Myers, who has satisfied himself by repeated investigation that the parasites show themselves first in the blood shortly after six o'clock in the evening, their numbers steadily increasing and reaching a maximum about midnight, after which a decrease sets in, and the swarm of filariæ disappears altogether between six and eight o'clock in the morning. These observations of Manson and Myers have been corroborated by Stephen Mackenzie³ in an ingenious experiment which he devised at the London Hospital in the case of a patient invalided home from India with hæmato-chyluria.

¹ 'Proc. Linn. Soc.,' 1878.

² 'Lancet,' 1878, f. c.

³ 'Path. Trans.,' xxxiii, 1882, p. 394.

During a period of some three months, when he was under observation, the urine passed by the patient contained sometimes a considerable amount of pure blood or of blood-clot, and sometimes it assumed a milky or chylous character; in the matters that were passed, and particularly in the blood-clot, *filariae* were detected, some of them being alive. It was a remarkable circumstance that, if there were any difference in quality between the day and night urine, it depended on the day urine having usually more of blood and fibrinous-clot and a larger number of *filariae*, while the night urine had a more chylous appearance. The examination of the blood, which was made every three hours for a space of two months, showed that it followed the converse rule as regards the presence or absence of *filariae*. During the day they could hardly be found at all in the blood; it was not until nine o'clock in the evening that they began to show; they were most numerous about midnight, decreased remarkably about three in the morning, at six were to be seen only now and then, and from nine in the morning to nine at night were absent altogether.¹ Changing the patient's meal-times had no effect whatsoever upon this behaviour of the parasite; but complete reversal of habit, to the extent of lying in bed during the day and being up and about through the night, was followed by a corresponding reversal in the periodical appearance and disappearance of the *filariae*, which showed themselves in the daytime and were to be seen only occasionally at night. A return to the patient's ordinary habits brought the parasite back to its original behaviour.

No explanation at all satisfying has hitherto been given by observers of this strange phenomenon, which reminds one of the life-history of *Oxyuris*. In regard to many other questions of *filaria*-disease the views of authorities are still widely divergent; for example, as regards *the true seat of the parasite*, whether it is both the blood-vessels and the lymph-vessels, as Lewis, Pereira, and others assume (with good reason, it seems to me), or whether, as Manson believes, it is exclusively the lymphatic system;² or, again, as to the ways in which it affects the human body, and as to the type of disease which ensues. Without attaching any special importance to the scepticism of Fox and Gouët, who would strike out altogether the term "*filaria*-disease" as the designation of definite forms of illness, inasmuch as they take the parasite to be a harmless guest presenting itself as a casual concomitant of diseases that have been brought about

¹ Havelburg has recently arrived at almost the same facts in a case of chyluria which he observed in Brazil.

² Havelburg also thinks that the lymphatic system is the part which is really implicated, the *filariae* passing from thence into the venous system.

by other etiological factors ; we may still hesitate before we go very far in the way of extending the application of the term. Numerous observations teach us that persons may be invaded by filariæ without suffering any appreciable injury to their health ; in many of the cases that have been published there is really no evidence that the sickness stood in any causal relation to the filariæ co-existing with it. It is beyond question, also, that cases of chyluria occur in extra-tropical regions, which are quite unconnected with *Filaria sanguinis* ;¹ and I take it, further, to be well-established that “elephantiasis” is a pathological term under which are included various kinds of morbid conditions differing from one another in their anatomy and in their etiology, the filaria-disease being only one of the number. However curious may be the evidence adduced by O'Neill and Arango that *Filaria sanguinis hominis* occurs in diseases of the skin, these observers are still under an obligation to prove to us that the skin disease which they saw does as a matter of fact correspond to the disease known on the West Coast of Africa as “craw-craw”—a disease which all authorities in that part of the world admit to be nothing else than scabies. I am very doubtful whether it was really the filaria in question that Neilly found in a case at Brest, of a vesicular pruriginous exanthem, closely related, as Neilly thought, to “craw-craw.”

§ 116. GEOGRAPHICAL DISTRIBUTION OF FILARIA SANGUINIS.

These brief references will suffice to indicate how much is still wanting to a perfect knowledge of all the circumstances concerned in the filaria disease ; and assuredly the same incompleteness characterises our information about its *geographical distribution*. The filaria-disease prevails most extensively in the tropical parts of *Brazil*, equally in the interior and on the coast.² In the provinces lying most to

¹ Havelburg (l. c., p. 375) thinks that in such cases the filariæ had perhaps been overlooked. His other supposition seems to me to be much more likely, namely, that there are other morbid processes which may lead to obstruction in the thoracic duct as the filariæ do, and which likewise produce chyluria.

² See the account in ‘*Revista med. flumin.* ;’ also Sigaud, Juvenot, Plagge, Wucherer, da Silva Lima, 1878, l. c., and Havelburg.

the south or in the higher latitudes, beginning with the province of Sta. Catarina, the disease in its hæmato-chyluric form is seldom met with. Neither is hæmaturia endemic in the Argentine Republic, according to Crevaux's¹ statement as against Juvenot's. How far the latter authority's account of endemic hæmaturia in *Chili*, *Peru*, *Venezuela* (Guaira), and *Mexico* is deserving of credit, can hardly be decided, as all other observers in these countries say nothing of the malady in question. For *Guiana* we have merely the above-mentioned notice by Winckel, who found the parasites in a woman long resident in Surinam, and Hillis's observations on a patient belonging to Demerara. As regards the *West Indies*, mention is made of the disease (hæmato-chyluria) in Cuba,² St. Domingo,³ St. Thomas,⁴ Barbadoes,⁵ Martinique,⁶ and Guadeloupe,⁷ where it occurs for the most part in merely occasional cases; it would appear, therefore, not to be widely diffused in these colonies. I am at a loss to decide whether the cases mentioned by O'Neill warrant us in concluding for the common occurrence of the disease on the *West Coast of Africa*; there is not a word about hæmaturia or chyluria in the accounts from that region, and it must remain doubtful for the present whether the elephantiasis that is endemic there depends upon *Filaria sanguinis*. The occurrence of the parasite in *Egypt* has been placed beyond doubt by Sonsino and Fayrer. Crevaux⁸ quotes a paper by MacAuliffe, which I am not acquainted with, to the effect that the people inhabiting the shores of the Zambesi and Lake Nyassa (*South Africa*) suffer from hæmaturia chylosa; it is certainly a question whether there has not been here some confusion with the distoma-hæmaturia which is endemic there (see p. 296). Reliable information on the filaria-disease comes from the *Zanzibar coast*,⁹ *Mauritius*,¹⁰ and *Réunion*;¹¹ and it is probable that the references by Grenet from *Mayotte* and by Vinson from *Madagascar* relate to the same disease. Of *Filaria sanguinis hominis* in *Queensland*

¹ 'Arch. de méd. nav.,' l. c.

² Beale.

³ Juvenot.

⁴ Pontoppidan.

⁵ Ralfe, Fayrer.

⁶ Rufz, St. Vel, Venturini.

⁷ Crevaux.

⁸ 'Arch. de méd. nav.,' l. c., 173.

⁹ Ferrand.

¹⁰ Chapotin, Salesse, Rayer.

¹¹ Quevenne, Mazaé-Azéma, Cassien, Pellissier.

we have Bancroft's account; but it is impossible to decide for the present whether the parasite is indigenous in other parts of the Australian continent. From *Oceania* there is intelligence of only one case hitherto, which Chassaniol and Guyot saw in an individual long resident in Tahiti.

Next to Brazil, *China* would appear to be the country most infested by filaria;¹ and it is remarkable that an island so near the mainland as *Formosa* is, according to Myers, quite free from it.² In *India* also, according to Lewis, McLeod, McCormack, Ewart, Carter, Barbour and others, the filaria-disease is very common. On the other hand, during a residence of many years in the *Dutch East Indies*, v. Leent and Swaving did not have the opportunity of seeing a single case of hæmaturia chylosa.⁴

Such is the extent of our information—it is not all equally trustworthy, and it is certainly very incomplete—as to the occurrence of *Filaria sanguinis hominis* in various parts of the world. According to our present knowledge, the parasite appears to be indigenous only in *tropical regions*; but within these it has been found in persons of all races and nationalities.

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¹ Manson, Siegfried.

² His own investigations were made on patients who had come there from the mainland.

³ According to a statement in Crevaux, 'l' Hématurie,' p. 28.

1872, Septbr. Crevaux, De l'hématurie chyleuse, etc. Par., 1872; Arch. de méd. nav., 1874, Septbr., 165; Journ. de l'Anat. et de physiol., 1875, 172.

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Salesse, Diss. sur l'hématurie, etc. Par., 1832. Dos Santos, Gac. med. da Bahia, 1877, Marte, 137, Novbr. Siegfried, Philad. Med. Times, 1878, Oct., ix, 4. Sigaud, Du climat et des malad. du Brésil. Par., 1844, 398. Silva Lima, Memoria sobre a hematuria chylosa. Bahia, 1876; Gac. med. da Bahia, 1877, Septbr., Nov. (Arch. de méd. nav., 1877, Decbr., 439; 1878, March, 200); Lancet, 1878, March, 441. Sonsino, Ricerche, etc. (cf. Distoma hæmat.); Rendiconto della reale Acad. di Napoli, 1874, Fasc. 6; Sugli ematozoi come contributi alla fauna entozoica egiziana. Cairo, 1877, 10; Lancet, 1882, May, 825.

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Winckel, Arch. für klin. Med., 1876, xvii, 303. Wucherer, Gac. med. da Bahia, 1868, Decbr., Nr. 57; 1869, Septbr., Nr. 77 to 79 (Zeitschr. für Parasitenkunde, 1869, i, 376). Arch. de méd. nav., 1870, Févr., 141.

Guinea-Worm (*Filaria medinensis*, *Dracunculus medinensis*).

§ 117. HISTORY OF THE PATHOLOGY OF DRACONTIASIS.

Although the *history of dracontiasis* can be followed far back into antiquity, it is to modern times that we owe the true knowledge of the nature of the disease; while the perfect comprehension of the life-history of the parasite which occasions it belongs to the recent period. Not to mention the indications of the dracunculus which Bartholin, and after him Küchenmeister, would find in the “fiery serpents” that afflicted the Hebrews in their wanderings in the Desert, the first definite facts about the disease that we meet with are in Plutarch,¹ who narrates, on the authority of a statement by the geographer Agatharchides, the teacher of Ptolemy Alexander (about 150 B.C.), that the dwellers by the Red Sea suffer from a serious malady, due to “a small serpent” (δρακόντια μικρά) which issues from the skin to gnaw the arms and legs and retires underneath the skin if disturbed, causing the patient intolerable pain. A subsequent notice of this worm occurs in Leonides² (second century of the Christian era), who compares it to the round-worm, and speaks of it as occurring in Ethiopia and India. Galen,³ who confesses that he had never had an opportunity of seeing a case of dracontiasis, expresses doubt whether it is really an affair of a living animal, and not rather an affection of the veins resembling varix; also Soranus (the methodist no doubt) had said:⁴ “Neque animal prorsus sed

¹ ‘Symposion,’ lib. viii, quæst. 9.

² In Actius, lib. xiv, cap. 86, ed. Basil, 1535, iii, 69.

³ ‘De locis affectis,’ lib. vi, cap. 3, ed. Kühn, viii, 392, and ‘Introductio,’ cap. 19, e. c., xvi, 790.

⁴ According to Paulus, lib. iv, cap. 59, ed. Lugd., 1551, 332.

nervosi cujusdam substantiam esse, quod opinionem motus solum praebeat.” These doubts and surmises were the beginning of an erroneous doctrine which dominated professional opinion in all subsequent times down even to the commencement of the present century. Even among the Arabian physicians,¹ who were well acquainted with the malady as it occurs in Arabia and Persia—Khorassan is specially mentioned by Avicenna—and had certainly had it under observation,² there was much vagueness as to its nature; still more so among the practitioners of the middle ages and first part of the modern period, who had no opportunities of seeing cases of dracontiasis, and relied for their belief solely upon their authorities, Galen and Avicenna. Although it was clearly proved by the unbiassed inquiries of a few investigators of the seventeenth and eighteenth centuries—such as Welsch,³ who overthrew Avicenna’s notion of the “vena medinensis” by a rigorous criticism of it, he himself trusting to observations made on persons who had come back to Europe from India affected with dracunculus,—and still more clearly through the experience brought from countries infested with the parasite by many scientifically trained practitioners and naturalists, such as Lind⁴ and Gallandat from the West Coast of Africa, Kämpfer from the shores of the Persian Gulf, Rouppe from Curaçao, Pouppé-Desportes and Peré from San Domingo—although it was thus proved that the disease was due to a living

¹ Abulcasem, ‘Method. med.,’ lib. ii, cap. 91, Basil, 1541, 162; Avicenna, Canon lib. iv, Fen iii, tract. ii, cap. 21, Venet., 1564, ii, 128; Haly Abbas, Lib. theor., viii, cap. 18, Lib. pract., ix, cap. 64, Lugd., 1523, 98 b., 283 b.; Avenzoar, Theisir, lib. ii, tract. vii, cap. 19, Venet., 1490, fol. 32 b.

² Abulcasem speaks of the parasitic nature of the malady in the following very definite terms:—“Vena haec generatur in cruribus, in terris calidis, sicut in terra arabum et orientalibus, meridionalibusque regionibus, terris aridis. Et quandoque generatur in locis aliis corporis praeter crura. Et generatio quidem ejus est a putrefactione quae accidit sub cute, sicut accidunt intra corpora serpentes, vermes, ascarides at vermes inter cutem et carnem.” He further mentions a case operated on by himself, in which he removed a “vena” twenty handbreaths long, or about sixty inches. As regards the term “vena,” I may remark that it is a purely arbitrary rendering of the Arabic term “irk” or “ark,” which was used in several senses.

³ ‘Exercitatio de vena medinensi, &c.,’ Aug. Vindel, 1674.

⁴ The writings of these and other authors quoted are given in alphabetical order at the end of the section.

animal within the human body ; and although the foremost men of science in Europe in the first quarter of the present century regarded the question as being so completely settled that Rudolphi¹ could say : “ *Nostris temporibus filariam istam in dubium vocari possit, fere omnem fidem superat ;*” yet it continued to be a subject of controversy whether the alleged worm was not altered connective tissue, or obliterated vessels, or a morbid condition of nerve,² until at length the more recent investigations in parasitology dispelled all doubts as to the parasitic nature of the disease.

§ 118. GEOGRAPHICAL DISTRIBUTION OF THE GUINEA WORM.

The *indigenous habitat* of *dracunculus* is a comparatively small area embracing a few regions of the Eastern Hemisphere, mostly intertropical ; and even there the parasite is found only within certain narrow limits. From these, its original seats, the worm has been imported to other lands ; but only in a few such foreign centres has it become domesticated.

One of the chief seats of *dracunculus* is the *West Coast of Africa* from the Senegal down to Cape Lopez. In the basin of the Senegal the parasite is common both on the level coast country and in the more elevated parts of *Senegambia* beyond Bakel to the region of Galam,³ although the banks of the Casamance enjoy immunity.⁴ In less extensive diffusion it occurs on the *Sierra Leone Coast*;⁵ but most extensively of all⁶ on the *Grain Coast, Ivory Coast, Gold Coast, and Slave Coast*,⁷ as well as on the shores of the *Niger*⁸ and the *Gaboon*,⁹

¹ ‘Entozoorum synopsis,’ Berl., 1819, p. 207.

² In the ‘Edin. Med. and Surg. Journ.,’ 1831, Jan., p. 112, Grant publishes an article written in 1830 by Surgeon-General Milne, of Bombay, whose conclusion from a study of a case of *dracunculus* is—“The substance in question cannot be a worm, because its situation, functions and properties are those of a lymphatic vessel ; and hence the idea of its being an animal is an absurdity.”

³ See Paré, Berville, Gauthier, Thaly, Bérenger-Féraud, Defaut, Hébert, Borius.

⁴ Léonard.

⁵ Boyle, Clarke (Sierra Leone).

⁶ See Gallandat, Lind, Reynhout, Boyle, Birkmeyer, Ref. in ‘Bost. Med. and Surg. Journ.,’ 1843, June, p. 293, Robinson, Bryson, Daniell, Heymann, Gordon, Clarke, in ‘Transact. Epid. Soc.’

⁷ Férís.

⁸ Oldfield.

⁹ Peré.

the more elevated countries inland such as the kingdom of Dahomey and the Tellatah territory being apparently quite exempt.¹ The points most infested are on the Gold Coast from Apollonia to the Rio Volta, including Cape Coast Castle,² Elmina,³ Cormantia, and Accra; but there are other places not many miles from these in which there is hardly a trace of the worm to be found.⁴ On the Bight of Biafra dracontiasis begins to be more rarely met with,⁵ and on the coast of Lower Guinea (Congo coast) the disease occurs very seldom if at all.⁶

In the medico-topographical accounts from the countries of the *North African* seaboard, the dracunculus is either not mentioned, or, as in the case of *Tunis*⁷ and *Egypt*,⁸ its endemic occurrence is expressly denied. In Egypt, as the authorities unanimously state, the parasite had been frequently observed after the conquest of the negro countries (Sennaar and Kordofan), both among the black soldiers brought thence, and among the Arabs, Egyptians and Europeans, who had been in the habit of mixing with the former. But, since the negroes are no longer employed in military service, dracontiasis is now met with only in such persons as had brought the parasite with them from its indigenous regions.

To the regions of Northern Africa in which dracontiasis is endemic, belong *Nubia*, *Kordofan* and *Darfur*,⁹ probably also some localities on the northern borders of the *Sahara*;¹⁰ while the *Greater Soudan*, so far as we know, is, like the Tellatah countries already mentioned, free from the disease.¹¹ In *Abyssinia*, dracunculus would appear to be found only on the coast.¹² For the East Coast of Africa, and adjacent

¹ Duncan.

² Clarke, in 'Transact. Epid. Soc.,' Moriarty.

³ Clymer.

⁴ Busk.

⁵ Daniell, Bryson.

⁶ Peré, Falkenstein.

⁷ Ferrini.

⁸ Clot-Bey, Fischer, Pruner, Bilharz, Vauvray.

⁹ Bruce, Marduchi, Fischer, Pruner, Mahomed-el-Tounsy, Hartmann.

¹⁰ According to the accounts by Ferrini for Tunis, and Bertherand for Tuzgurt (Algiers); see also Richardson, l. c.

¹¹ Tutschek, Quintin.

¹² Harris, Hartmann, Currie.

islands,¹ as well as for the Cape, Australia² and Oceania, there is no mention of the parasite.

The endemic seats of dracontiasis on *Asiatic* soil, are first of all *Arabia Petraea* (wrongly called "the stony"), a few points on the coast of Hejaz and Yemen,³ and the south coast of *Persia*.⁴ In other parts of Persia, such as Teheran, the disease is seen only in persons who have come from a distance.⁵ From *Syria* there is a statement by Nathan, to the effect that a number of sailors of the English navy, who had never been in tropical or other countries infested by *dracunculus*, contracted the parasite after working in the water for a considerable time in the Bay of Skanderoon. Even in the northern regions of Nearer Asia, there are isolated centres of the disease to be met with, particularly in some parts of *Turkestan*, in Khiva, Bokhara⁶ (limited to the city of that name) and Kokaun;⁷ also along the shores of the Sir-Daria (*Kirghiz Steppe*),⁸ and even on the northern shore of the *Caspian* in latitude 47° N., "prope flumen Paccum," as Kämpfer says, meaning probably the banks of the Jaek or Ural.

Next to the West Coast of Africa and the upper basin of the Nile, it is in India that the guinea-worm attains its widest diffusion, and mostly in the northern division of the West Coast, the Rajpootana States and the western parts of the Deccan. Among the least infested parts of the country, according to Balfour, are the *North-West Provinces*, whence there are accounts of dracontiasis (known to me) only from Dehra Dhun⁹ (30° N., 95° E.), Sirsa¹⁰ (29° 31' N., 92° 45' E., in the district of Bhatti), and Hansi¹¹ (29° 6' N., 93° 43' E., in the district of Hissar); also those parts of

¹ According to Collier and Paton, dracontiasis does not occur in Mauritius and St. Helena.

² Thomson says the same of New Zealand.

³ Clot-Bey, Fischer, Pruner, Harris, Bilharz. There appears to be little reason for calling the worm "*Filaria medinensis*," for it is of rare occurrence in Medina, as also at Jeddah.

⁴ Kämpfer, Pruner, Busk, Moore, Polack, Leblanc.

⁵ Polack.—Avicenna, whose opinion in this matter must be judged correct, speaks of the endemic prevalence of the malady in Khorassan.

⁶ Mir-Izzet-Ullah, Burnes.

⁷ Fedschenko.

⁸ Maydell.

⁹ Brett.

¹⁰ Minas.

¹¹ Balfour.

Bengal situated in the lower basin of the Ganges, whose immunity is indicated not only by the unanimous statements of Balfour, Greenhow, Twining, Voigt, and others, but also by the absolute silence as to this disease among the extremely copious medical writings relating to Bengal and Orissa. In the coast belts of the *Madras* Presidency also (Northern Circars, Carnatic and Cochin), dracunculus is of comparatively rare occurrence,¹ being found only at isolated places, such as the vicinity of Madras² and Pondicherry.³

According to all observers,⁴ the name of the disease is scarcely known on the eastern and western Ghâts of this Presidency, and as little on the high ground of Mysore. The single more considerable focus of the malady in this part of India is the *plain of the Carnatic* stretching southwards from Mysore between the eastern and the western Ghâts towards Cape Comorin, in which Dindigul,⁵ the Salem district,⁶ Madura, and many other places⁷ at a distance of one or two days' journey from the coast, are given as the seats of dracontiasis. But the greatest prevalence of the disease is on the western seaboard at the following places: in the *Bombay* Presidency, from the latitude of 18° N. up to Gujerat,⁸—at Rutnagherry, Matunga, Bombay (but not at Kolaba only a few miles from the capital)⁹ and Daman; in *Gujerat*,¹⁰ at Baroda, Caira and Jambosir; and at Bhooj, in Cutch.¹¹ Other great centres of the disease are met with in the *Rajpootana* States (Mewar and Marwar),¹² in the district of *Chanda*,¹³ at Dhoolia in *Kandeish*,¹⁴ at Nagpore in *Berar*, in the *States of the Nizam*,¹⁵ as at Aurungabad, Jalnah, Hyderabad and Secunderabad, on the east side of the western Ghâts, and in the adjoining districts of the *Deccan*, from which last we have accounts of the prevalence of dracunculus at Ahmednuggar, Jedjhuri, Baramati, Poona,

¹ Scot, Day.² MacKay.³ Huillet.⁴ Dubois, Lorinser, Gibson, &c.⁵ Annesley.⁶ Cornish.⁷ Dubois.⁸ Morehead, Duncan, Ewart, McGregor, Scott, Bird, Carter, Crespigny.⁹ McGregor.¹⁰ Gibson.¹¹ Moore.¹² Ewart, Greenhow, Moore.¹³ Dutt.¹⁴ Mackenzie.¹⁵ Lorinser, Morehead, Cooper.

Satara, Aculcota, Tasgoon, Miraj and Beejapore,¹ the district of Savant-Warri,² Balgâm,³ Darwar⁴ and Bellary.⁵

A means of estimating the relative frequency of the disease in the Presidencies of Bombay and Madras respectively, is afforded by Ewart's figures. In Madras from 1829 to 1839, there was one case of dracunculus to 562 native troops, and among the European troops one in 1800; in Bombay during the same period one in 32. At some of the military stations in the Bombay Presidency, according to Morehead, the rate of sickness from dracunculus was 12 to 17 per cent. of the total strength.

If we may judge from the silence of the authorities, dracunculus does not occur at all in *Ceylon*, *Further India*, *China* and *Japan*. From the *East Indies* we have information by Heymann, van Leent and v. d. Berg, that dracontiasis was quite unknown in Java before the arrival of African troops, that the parasite was imported by them from Elmina, that it has disappeared again since these military transfers ceased, but that even at present the disease may be seen now and then in Africans, or in such of the Europeans as had lived for a considerable time on the West Coast of Africa, but no cases of it in the Javanese or other Asiatics who had not left the East Indies.

According to the unanimous opinion of the medical authorities for *Guiana*,⁶ *Brazil*⁷ and the *West Indies*,⁸ the dracunculus was imported into these countries of the New World by negroes from the West Coast of Africa; and it has almost disappeared again from them, excepting at one or two small centres, since the importation of negroes has ceased. One of these centres is the island of Curaçao, into which, as Rouppe tells us, the disease was brought as usual by negroes, and in which it is said that there are still cases of dracontiasis occurring somewhat frequently among the native population.⁹ The other centre is the small village

¹ Morehead, Collier, Gibson.

² Kearney. ³ Waller.

⁴ Forbes.

⁵ Eyre.

⁶ Rodschied for British Guiana, Bajon for Cayenne, Schüller and Hille for Surinam.

⁷ Sigaud, Schwarz.

⁸ Peré and Pouppé-Desportes for St. Domingo, Sloane for Jamaica, Savarésy for Martinique, Hillary for Barbadoes.

⁹ Busk. Popin writing from Curaçao ('Nederl. Tijdschr. voor Geneesk,' 1859, iii, 214) makes no mention of the disease.

of Feira da Santa Anna in the Province of *Bahia*, between the town of Bahia and Jazeiro, where there is an endemic focus, according to statements (which we shall recur to, p. 354) by Pereira and da Silva Lima. Whether the parasite is indigenous there, as the latter authority thinks, or whether it has been imported, as Pereira believes, we are unable to decide. For the remaining countries of South America (the River Plate States, Chili, Peru, Bolivia, Ecuador and Granada) as well as for Central and North America, there is not a single reference to the endemic occurrence of dracontiasis.

§ 119. RELATION OF DRACONTIASIS TO HEAT AND MOISTURE.

The limitation of the area of dracontiasis almost exclusively to regions situated within the tropics raises the suggestion that the existence of the parasite which causes the malady is dependent upon *circumstances of climate*, or, in other words, upon *high temperature*. No doubt the endemic occurrence of the disease in Turkestan, and on the Kirghiz Steppe, where the mean temperature of the year is scarcely higher than that of the South of Europe, appears to contradict this assumption. But in the problem before us we are concerned not with the isotherm but with the isother; and, in that respect, the regions in question have a truly tropical climate, inasmuch as their mean temperature in summer is 25° R. or more (88° Fahr.); and, what is especially noteworthy, it is precisely the hot season, notorious for its extreme drought, that forms the proper filaria-season, during which, as Burnes states in regard to the city of Bokhara, one eighth of the population suffer from dracontiasis. Moreover, the influence of the hot season on the prevalence of the disease makes itself unmistakeably felt in tropical countries themselves. In almost all the territories infested with dracontiasis, for example, Senegambia,¹ the West Coast of Africa,² Nubia,³ and the various Indian centres (Rajpootana,⁴ Bombay,⁵ and Madras⁶), the largest number of cases

¹ Gauthier, Borius, Hébert.

² Reynhout, Robinson, Gordon, Clarke.

³ Clot-Bey, Fischer, Pruner, Petherik.

⁴ Moore (III), Greenhow.

⁵ Bird.

⁶ Lorinser.

falls in the rainy season or in the hot season following it, that is to say, in April and May, or from June to August—September, according to the locality.

On this point we have two considerable series of observations, one by Ewart for Mewar, and the other by Morehead for Bombay. Among the troops composing the Mewar Bheel contingent there occurred, according to Ewart, 2682 cases of medina worm during a period of 17 years; and these were distributed over the months of the year as follows:

September	103	March	239
October	96	April	420
November	57	May	525
December	29	June	493
January	23	July	376
February	65	August	256
	<hr/>		<hr/>
	373		2309

According to these figures the ratio of cases in the second column (March to August) was to those in the first (September to February) as 6 to 1. In Morehead's statistics of 2927 cases of *dracunculus* admitted into the Bombay hospitals, the distribution throughout the year was as follows:

October	224	April	273
November	123	May	448
December	93	June	480
January	46	July	428
February	64	August	337
March	165	September	246
	<hr/>		<hr/>
	715		2212

This gives a proportion of 3·2 to 1. In both places the maximum falls in May and June, amounting in the one series to 1018, or nearly 38 per cent. of the whole, and in the other to 928, or 32 per cent.

It is impossible to decide absolutely to what extent the high temperature of the hot season by itself determines the prevalence of the disease (or infection by the worm) or how far the antecedent or accompanying *heavy rains* may be associated with it, as some observers are inclined to think. There are no certain data of the amount of rainfall in each preceding year (during which the infection must have taken place, since the period of development of the parasite lasts from nine to twelve months) to place beside the corresponding numerical return of the number of cases; and the absence of exact

meteorological facts of that kind is most of all to be regretted on account of those instances where the *dracontiasis* has broken out as an epidemic. These epidemics have been observed on several occasions—by Ewart in Mewar, by Eyre in Bellary, by Leblanc in Persia, by Ferg in Surinam, by Forbes in Darwar, and by Morehead in Kirkee. Some of these authorities would explain them by the fact that there was an unusually heavy rainfall in the previous year, although they do not support that statement by figures, nor adduce evidence that there is ordinarily a tolerably constant relation between the rainfall and the amount of the disease at the particular place. The only statement of a more precise kind that I have met with is one by Ewart, and it does not tell in favour of this theory. The largest number of cases in the period from 1841 to 1858 among the Mewar Bheel contingent, he says, happened in the year 1858, although the amount of the rainfall the year before was within the mean annual range; the smallest number of cases was in 1855, although the rainfall of 1854 exceeded the annual mean by a good deal (29 inches), and, conversely, when in 1853 the rainfall was as much as 18·4 inches below the annual mean, the cases of dracontiasis that came to be treated in the year 1854 were comparatively few. Moreover, it is the opinion of several authorities, among others Robinson for the Gold Coast, Annesley for the Carnatic, and Lorinser for Secunderabad, that hot and dry weather is particularly favorable to the infection; and it is a not uncommon belief in India, as Ewart tells us, that the frequency of the disease in any year stands in an inverse ratio to the amount of the rainfall the year before.

§ 120. ALLEGED PREFERENCE OF DRACUNCULUS FOR CERTAIN SOILS.

Still more questionable than the rainfall, as an influence upon the amount of the disease (in other words, on the abundance of the parasites and the opportunities for infection by them), are certain *conditions of soil*, to which some observers would attach significance in this respect. Chisholm

was the first, so far as I know, to attach special importance to a volcanic soil for the occurrence of dracontiasis, according to his own observations in Grenada. In the same sense Morehead afterwards asserted that the malady was endemic on the western littoral of Hindostan and in the Deccan, nowhere but at those places where basalt (basaltic tufa-trap or the so-called "mohrum") was the prevailing formation; and that it did not occur on primary rock, laterite, diluvium, or other formations. In particular he pointed out that as far as the conglomerate rock, consisting mostly of ferruginous clay (laterite) extended along the coast, that is to say, from Cape Comorin to the mouth of the Bankota, the country was either absolutely free from the malady or was affected by it very slightly; whereas the proper region of *dracunculus* is reached when the trap rock crops out to extend along the coast northwards. These statements have been confirmed by Day and Gibson. Waring also has expressed the opinion that the disease is much more common on volcanic trap than on a lateritious soil; and in a like sense Stewart has pointed to the notable exemption from dracontiasis enjoyed by Jubbulpore, situated on laterite, in the Nerbudda district, while the disease is endemic on sandy soil in the vicinity of the town. Horton also has brought forward the prevalence of *dracunculus* on the volcanic trap or on the metamorphic rock of the Gold Coast.

I shall not attempt to decide whether volcanic rock is in any degree favorable to the prevalence (or the existence) of the guinea-worm; but it is quite erroneous to suppose that laterite prevents it. And if we look at the kinds of soil upon which the disease is endemic in other parts of the world it becomes extremely questionable whether the geological character of the ground has any influence whatsoever upon the occurrence of dracontiasis, that is to say, of the *dracunculus* parasite.

In Mewar and other parts of the Rajpoot States which are affected with dracontiasis, the soil is of the oldest formations (granite, gneiss, red sandstone and the like). In the plain of the Carnatic, where we have seen that there is a very extensive focus of the disease, we find primitive rocks and clay slate. Those parts of Nubia where the malady is most endemic rest upon sandstone covered by a layer of calcareous tufa

(belonging to more recent formations), or by a considerable stratum of clayey or calcareous diluvium, or in parts covered by laterite. In Bakel (Senegambia) we meet with dracunculus on calcareous clay slate. At many points on the Guinea Coast where dracontiasis is most frequent, the soil is of laterite and is covered at the estuaries of the Niger and other large rivers with more or less of ferruginous diluvial clay washed down from the higher regions; whereas at other points where volcanic and particularly basaltic formations are most prominent, such as the Sierra Leone coast, the disease is of the rarest occurrence, and on the Congo coast is quite unknown. Bokhara, which is severely infested by the parasite, rests on a soil of loam impregnated with salt.

§ 121. NO RACIAL, SOCIAL, OR OTHER IMMUNITY FROM DRACONTIASIS.

Dracontiasis has been found among *all races* and *nationalities*, in *all classes of society*, at *all periods of life*, and in *both sexes*. No doubt the Europeans suffer less than the negroes or other indigenous inhabitants of the affected regions; and cases are rarer among persons in good circumstances than among the working class, the common soldiers and such like; rarer also in the female sex than in the male. But the sole reason is that the one are exposed less to the infection, or take more care to avoid it than the other. The more frequent, however, are the opportunities of infection, the more do these differences appear to be equalised. Thus in the Presidency of Madras from 1829 to 1839, there was one case to 562 men for the whole of the native troops, but only one to 1880 for the European troops; on the other hand, in the severely affected Presidency of Bombay, the rate of sickness in the years 1832 and 1833 was almost the same in the two classes of soldiers, being about 3 per cent. of the total strength.

§ 122. LARVAL WORMS IN WATER CORRESPONDING TO GUINEA-WORM.

The *mode of infection* of the human body, or the *manner of invasion of the parasite*, has been the subject of erroneous

or at all events vague notions until quite recently, when it was thoroughly cleared up by the researches of Fedschenko in Turkestan. It had been long recognised and clearly stated by many scientific travellers and practitioners who had had opportunities of observing the medina-worm in its habitats, that it did not arise in the human body *de novo*, as was still believed at the beginning of the century by those who applied the doctrine of spontaneous generation to the case of the intestinal parasites; but that it invaded the body from without, not indeed in the mature form, but as a larva. Another opinion, firmly held by most of the natives of the infected countries, that the infection is caused by drinking the water of certain wells, pools, tanks, or sluggish water-courses,¹ has not only been countenanced by scientific observers,² but has been apparently corroborated by some of them when they showed from the examination of the suspected water, or of the damp or marshy ground near it, that these contained filaria-like worms often in enormous numbers, which resembled dracunculus or its larva, and might therefore be regarded as the offspring of that parasite.

The first published observations of that kind, so far as I know, were those of Chisholm. In his account³ of the water of certain wells in Grenada, which were notorious as the source of the filaria disease, he says: "In the water, which contains the embryos of the dracunculi, the naked eye distinguishes innumerable animalcules, darting in every direction with astonishing force and rapidity; these, on being subjected to examination in a small microscope, exhibit a very extraordinary figure, differing from any animalcules hitherto described."

¹ "In all countries," says Chisholm ('Edinb. Journ.,' l. c., 150), "in which the Dracunculus is endemic, the prevailing belief of the people is, that it proceeds from drinking water which contains the ova or the embryo of the animal."

² Thus, in Bruce's 'Travels' it is stated that the worm usually attacks those persons whose practice it is to drink stagnant water. Chevalier also, in his letters on the diseases of San Domingo, says: "Ces vers sont engendrés dans le corps par les mauvaises-eaux;" Kämpfer and other travellers expressing themselves to the same effect. Among practitioners, again, there has been only one opinion,—that the parasite, or its eggs or larvæ, come to the human body out of water, their views diverging only as to the mode of entrance.

³ First published in the 2nd ed. of his "Essay on the Boullam Pestilential Fever in the West Indian Islands, 1793-96," 2 vols., i, 571, note, 1801, and afterwards in 'Edin. Journ.,' l. c., p. 150.

Duncan, who found on microscopic examination of the mature female that the uterus was packed full of embryos ("the white matter filling the inner tube like thick cream is a mass of living young ones"), goes on to say that "the soil and pools abound in the rains with a worm smaller and more slender, but otherwise exceedingly like Nharroo"—the colloquial name for *dracunculus* in India. Forbes, writing of Darwar says: "I examined several of the tanks in the neighbourhood and found the mud on their banks, and in their half dry beds, abundantly supplied with animalcules, some of them very much resembling those produced by the guinea-worm when infesting the human limb Two kinds of these animalcules may be detected in the soft mud: one kind seven or eight times the size of the guinea-worm animalcule, the other exactly resembling it."

Brett says that he has found *dracunculus* in flood-pools on the banks of the river Dhun, notably at Dehra-Dhun; and a writer from the Gold Coast¹ alleges that he saw in rain-water taken from a cistern two *filariæ* which proved on microscopic examination to be the real *Filaria medinensis*. Carter, also, expressed a very definite opinion on the identity with *dracunculus* of a certain species of *filaria* which he found in tanks (and named accordingly "tank-worms"); and this view was afterwards accepted by Schwarz, who had opportunities during a residence in Bombay of comparing the embryos of *dracunculus* with the tank-worms in question. Ewart also adopted that opinion, pointing out, as Carter had already done, that these parasites were never found in pure water, and that those persons who used nothing but pure water, were never affected with *dracunculus*.

§ 123. EVIDENCE FOR AND AGAINST THE ENTRANCE OF DRACUNCULUS WITH THE DRINKING-WATER AND THROUGH THE SKIN RESPECTIVELY.

I do not attempt to decide how much or how little credit we should give to these observations, or to some of them,

¹ See 'Boston Med. and Surg. Journ.,' 1843, June, p. 293.

Carter's in particular.¹ They served, at all events, to confirm the opinion that the invasion of the human body by the parasite took place in the larval form out of water; and the attention was then mostly directed towards answering the question, *by what door the parasite entered*, whether by the digestive organs (in drinking-water), as had been almost universally assumed according to the notices above given, or through the skin.

The objections taken to the drinking-water theory by many observers are supported partly on negative and partly on positive grounds. Not to mention the purely hypothetical exception that has been taken to it from a physiological point of view (by Clymer), the fact has been dwelt upon that there are places infested by the parasite where natives and European take their drinking-water from the same source, and yet the former suffer from *dracunculus* to a much greater extent than the latter, who not unfrequently indeed escape it altogether.

But the consideration that has special weight with the opponents of the drinking-water theory is that the worm, in the great majority of cases, has its seat in the subcutaneous connective tissues, particularly of the feet; and accordingly in those parts of the body that are farthest from the supposed channels of invasion.

Of 133 cases observed by Lorinser, 80 had the parasite located in the skin of the foot or ankle, 39 in the leg, 6 in the thigh, 2 in the scrotum, and 5 in the forearm. Of 210 cases given by Ewart, 120 had the filaria in the foot and ankle, 67 in the leg, 5 in the thigh, 2 in the scrotum and thigh, 2 in the abdominal wall and thigh, 2 in the forearm, 2 in the back and thigh, 7 in the knee and forearm, and 1 each in the thigh, abdominal wall and back, in the thigh and penis, and in the scrotum, perineum and groin. Of 369 cases treated by Grierson, 335 had the parasite located in the lower extremities, 29 in the upper, and 5 on the trunk.

This is certainly a very remarkable fact; and it may easily lead us to think that the guinea-worm comes to the body from the soil or water, not by imbibition, but through the skin (pores or sweat-glands). Rouppe had formerly inclined towards that opinion "*quia [dracunculi] eos qui nudis*

¹ Compare Leuckart's not very favorable verdict on Carter's doctrine ('Parasiten des Menschen,' ii, 703).

pedibus incedunt prae aliis plus invadant ;” and the same fact has served to determine the views of many observers in Nubia,¹ on the Guinea Coast,² and in India,³ who pointed out that the malady occurred chiefly among those who went about on the damp marshy ground, just as the natives do in the tropics, or waded in swamps, or had to work in standing water, or who bathed in the same ; and thus Bryson says of the filariæ, by way of summing up, that “ they are generally found in those parts that are most exposed to the influence of external objects.”

There has been, moreover, no lack of individual observations which seemed to give support to this theory. Heath observes, in his account of an outbreak of dracontiasis among the crew of a ship which had lain a long time in the port of Bombay, that the officers were engaged on shore just as much as the crew and drank the same water ; but the crew when employed on shore wore nothing but shirt and trowsers, whereas the officers, who escaped the disease, were completely clad and were protected by boots or shoes against the invasion of the filaria. Clarke relates that the troops in the barracks at Cape Coast Castle, in 1858, slept on mats on the ground, and suffered much from dracontiasis ; but the cases became considerably less common after beds were erected. Busk says that cases of guinea-worm have occurred on the West Coast of Africa in Europeans who had never been ashore, but had merely exposed some uncovered part of the body to the water in the boats of the natives. Interest was specially excited by the statement of Bruce⁴ that the “ bheesties ” or water-carriers in India, who carry the water in a leathern bag depending from the shoulders over the back and flanks, suffered from *dracunculus* chiefly in those parts of the body which came into direct contact with the bag ; and that statement was afterwards confirmed by Scott.

Plausible as this theory has always seemed, and energetically as Carter has lately entered the lists for it ; still the great majority of observers, relying on unambiguous experi-

¹ Clot-Bey, Fischer, Pruner, Petherik.

² Bryson, Bush, Clarke in ‘ *Epidemiol. Transact.*,’ l. c.

³ Bruce, ‘ *Edinb. Journ.*,’ l. c., Bird, Eyre, Gibson, Heath, Mackenzie, Carter, &c.

⁴ ‘ *Edin. Med. and Surg. Journ.*,’ l. c., p. 147 (note 1).

ences, have remained true to the original idea of the parasite being introduced with the drinking-water. The argument for entrance through the skin, which is derived from the localisation of the parasite in the subcutaneous connective tissue of the lower extremity, has been decisively met by Ewart when he points to the distant wanderings of many other parasites within the human body, and to their various favourite seats in some particular organ or tissue far removed from the point of invasion (echinococcus of the liver, cysticercus of the connective tissue, trichina of muscle, &c.), although in these cases there could not be the smallest doubt that they entered by the digestive canal. Again, it has been shown that dracunculus has occurred in a very large number of cases where there had been no exposure to infection through the skin, but on the other hand, particular care taken to avoid it—cases of persons belonging to the better-off classes, such as officers, civilian officials, and the like.

The statements of Bruce and Scott as to the occurrence of the worm in Indian water-carriers at those parts of the body which come into direct contact with the water-bags, have been rejected by Smyttan, Morehead, and Ewart as absolutely without foundation :

“I have never observed,” says Smyttan, “that Beesties most frequently have the guinea-worm in those parts which come in contact with the leathern water-bag, nor does it appear to me, that they are in any degree more subject to them than other descriptions of people;” and Morehead confirms this as follows: “I can affirm, after ample opportunity, and no little attention bestowed on the study of dracunculus, that I am unable to bring to my recollection a single instance of a water-carrier affected with it at that part on which the water-bag rests, nor have I any reason for supposing that they suffer more than other classes.”

But the leading argument in favour of the access of the parasite by the drinking-water is furnished by the instances where dracontiasis has broken out endemically or epidemically under circumstances which leave no doubt as to the mode of infection. Chisholm had long ago satisfied himself that the malady occurred on the Grenada plantations only among

those negroes who used to drink from the pools of water that had been partly filled by the tide and were therefore brackish ; whereas the negroes who used rain-water remained exempt, and the disease vanished from the plantations altogether when springs beyond the influence of the tide came into use, or when bricked cisterns were erected. Dubois's experience was the same as regards the distribution of *dracunculus* among the natives on the banks of the Cauvery (Southern India). Ferg observed in 1801 on a coffee plantation (Beninenburg) in Surinam, an epidemic of *filaria* in which nearly 200 negroes became affected within a space of four or five months, and not only the field hands, but also the slaves employed in the house of the manager, who had nothing in common with the others except the water supply ; and the inquiry that was set on foot showed that it was the use of drinking-water from the spring which had been the true cause of the outbreak. In Secunderabad, according to Cooper, an epidemic of *filaria* occurred among the native troops, which was almost exclusively confined to two companies that had got their water supply from a particular well. Dutt had under treatment 180 cases of *dracontiasis* from a village near Warora (Central Provinces), all of whom had got their drinking-water from a filthy well, while other inhabitants of the village, who had used a different water, continued free from the disease. A fact of great interest in this connexion is the outbreak of *dracontiasis* in 1849 among the members of two trading caravans who encamped on the road from Bahia to Jazeiro beside a stream a few leagues distant from Feira de Santa Anna ; disregarding the warnings of the natives, they made use of the water for drinking, although it is expressly stated that they did not bathe in it ; and a few months later all the members of the party fell ill with the exception of a negro, who was the only one of their number that had not drank the water. Moreover, the same consequences had followed, according to a practitioner living at Feira de Santa Anna, in the case of fifty of the inhabitants of a commune some six miles from that place, and among these there were several who had not bathed in the stream but merely drank the water.

The question as to the mode of entrance of the guinea-

worm into the human body has recently been brought near a solution, if not finally solved, by the researches of Fedschenko. That observer has shown¹ that the embryos of *dracunculus* enter the body of the small cyclops, which abounds everywhere, and develop in the interior of the latter into a larval form which had not been recognised before; that this invasion by the embryos takes place on the ventral surface of the cyclops; and that the development to the larval state within the intermediate host takes place usually above the intestine in the dorsal half of the body-cavity.² For the present we have no positive facts as to the later stages of the process; but it is a probable conjecture that the infection of the human body takes place through the cyclopeans which contain the larval parasite being taken in along with the drinking-water, the larvæ attaining their freedom in the stomach of man, starting on their travels, and undergoing development into the mature animal in a tissue of the human body adapted thereto. The infection of the cyclopeans by filarial embryos presupposes, naturally, that the latter find their way into water where their hosts live; but as Leuckart justly remarks, such opportunities can hardly ever be wanting where the worm is at all common. Moreover, this process enables us to understand the fact already mentioned, that *dracunculus* occurs mostly at places where there are the conditions most suited to the existence of the cyclopeans, namely sluggish, shallow or stagnant waters, pools abounding in vegetation, tanks, swamps, and the like; and in that respect also it will appear that there is justification for Ewart's conjecture that the decline in the cases of *dracontiasis* during the last ten years of his observations in the Mewar Bheel contingent is to be attributed to improvement of the water supply.

In the period from 1841 to 1849, the number of cases among these troops amounted to 18·24 per cent. of the total strength, while from 1849 to 1858 it fell to 13·93 per cent. This is to be explained, says Ewart, through the growing conviction that the malady was of most frequent occurrence among those persons in the cantonments who

¹ I follow the account of Leuckart (l. c., p. 704), as the language of the original paper is not familiar to me.

² For further particulars of the larval development see Leuckart, l. c.

were "the filthiest drinkers." Being convinced of this, the more intelligent of the men had used greater care of recent years in choosing what wells they would drink from. The European families resident there enjoy perfect immunity from *dracunculus*, the reason being, as Ewart is convinced, that they take the greatest care about their water-supply; and even among such of the native civilian population as take their drinking-water exclusively from wells that are kept clean, not a single case has occurred.

§ 124. GUINEA-WORM HAS FOLLOWED THE AFRICAN NEGRO TO AMERICA AND ELSEWHERE.

All the authorities for Brazil, Guiana, the West Indies, Egypt, and the East Indies agree in stating that *dracontiasis* was quite unknown in these countries before the importation of the negro; and in the subsequent period it has been always the African part of the population that has supplied the largest contingent of the sick. With the suppression of the slave trade, or, in other words, the cessation of frequent and extensive intercourse between the countries of the negro (especially the Guinea Coast) and the countries above named, the disease in most of the latter fell to a minimum or disappeared altogether. There can be hardly any doubt, therefore, about the *transmissibility of dracontiasis* or of the parasite which underlies it; and the interesting researches of Fedschenko supply the information as to the manner of that transmission. At the same time it follows from the experience of these countries, that the parasite is able to survive and to become domesticated outside its native habitat only under certain conditions; but the nature of these conditions cannot be in any way inferred either from the older observations or from the results of Fedschenko's inquiries. In like manner, for all those places in the *Eastern Hemisphere* where *dracontiasis* is now endemic, the question remains undecided, whether they were the *original habitats of the parasite*, or whether some of them, affording the conditions necessary to the existence of the guinea-worm, may not have been secondarily infected. For India in particular, this question may be urged. According to Gramberg,¹ the

¹ Quoted by Leuckart from the 'Geneesk. Tijdschr. voor Nederl. Indie,' 1861, ix, p. 632.

disease was imported by negro troops into Bombay, which is now the head-quarters of it in India. From Madras,¹ Waring gives the following example of the formation of new endemic centres: Previous to 1834 the city, and in particular certain of its suburbs, enjoyed almost complete immunity from dracontiasis. In 1834 and 1835 cases occurred here and there, and since that time the disease has gained so much in extent that it must now be reckoned among the maladies of most frequent occurrence. While the number of cases from 1834 to 1838 amounted to 222 in all, they rose in 1839-43 to 387, and in 1844-48 to 920. "I do not think," says Waring, "that we can come to any other conclusions than that, whatever may be the cause of the disease, it may be transplanted from place to place, and that localities previously exempt may become the seat of the affection."

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*Insects.*¹§ 125. GEOGRAPHICAL DISTRIBUTION OF SARCOPTES SCABIEI—
THE ITCH INSECT.

Historically and geographically considered, this parasite is coextensive with the human race itself.² Among the parasitic diseases that have been known in every age and in all parts of the world, the itch takes the foremost place. It has not rarely assumed the character of an epidemic under such unhygienic influences as specially favour the spreading and lodging of the parasite;³ not rarely also it has gained and maintained an endemic sway over the inhabitants of those countries or districts where ignorance, rough manners and the indolence springing from them have had the effect of keeping the social habits down at a primitive level.

We have no definite figures whereby to estimate the extent to which the itch is endemic in various parts of the world, the prevalence of the malady being denoted merely by such general terms as “rare,” “common,” “very common,” or “generally diffused.” But according to that kind of information, and so far as it goes, the portions of European soil most infested by the itch are *Iceland*,⁴ the *Faröe Islands*,⁵ *Norway*, *Poland*, *Russia*, some parts of *Eastern Germany* and of *France*, *Turkey* and the *Ionian Islands*.

¹ As I mentioned at the beginning of this chapter, I intend to consider here only those insects which have the character of true parasitic feeders upon man, and are at the same time among the more common or endemic of their class.

² Hebra has published a very exhaustive inquiry into the history of the disease from remote antiquity down to the present time (“Acute Exantheme und Hautkrankheiten,” in ‘Virchow’s Handb. der spec. Pathol. und Therap.,’ Erlangen, 1860, i, 410).

³ In the Napoleonic wars the sufferers from the itch in the French armies were counted by the hundred thousand. In the hospitals of Prague in 1866, according to Peters (‘Prager Vierteljahrschr. für Heilkde.,’ 1868, iv, 179; 1874, ii, 1), the number of patients admitted with the itch was 1129, while in 1867 it was 2256; in the years following there was again a decrease proportionate to the rise. No doubt the state of war in 1866 made the difference there also.

⁴ Schleisner, ‘Island undersögt, &c.,’ 26; Finsen, ‘Jagttagelser, &c.,’ Kjöb., 1874, 137.

⁵ Manicus, ‘Bibl. for Laeger,’ 1824, 15.

In Iceland, according to Finsen, the disease would appear to be rarer now than formerly. As regards France, Lanquetin¹ says: "Il y a encore dans le Jura plusieurs villages, dont les habitants sont à peu près tous galeux." And Hardy,² referring to Lower Brittany, says: "La gale s'empare du sujet quelques jours après sa naissance, le suit dans toute sa carrière et ne l'abandonne qu'à la mort." In the island of Cephalonia, according to Robertson,³ one third of the whole 60,000 inhabitants had the itch.

On Asiatic soil, the parasite is universally diffused in *Siberia*⁴ and *Kamschatka*,⁵ as well as in *China*⁶ and in *Japan*⁷, where, as Vidal (following Siebold) tells us, three-fourths of the whole population have the itch; and, as Friedel declares, not only the natives but also foreigners are affected. It is common also in the *East Indies*,⁸ where van Leent says of it: "La gale est tellement répandue parmi les indigènes, que beaucoup d'individus n'y portent même plus attention," adding that this holds good not only for the lower classes but also for people in better circumstances. Almost all the authorities for *India*⁹ mention the enormous frequency of this parasite among the native population: "On peut dire," says Huillet, "que presque tous ceux [galeux] de la basse classe vivent avec elle [la gale] sans chercher à s'en guérir." The same is true of *Arabia*¹⁰ and of *Persia*, where, according to Polak,¹¹ one half of the soldiers in a regiment arrived at Teheran from Tabriz, had the itch. A remarkable exemption is enjoyed by some of the island-

¹ 'Notice sur la gale,' Par., 1859, 63.

² 'Leçons sur les maladies de la peau,' Par., 1859, ii, 141.

³ 'Lond. Med. Repository,' 1818, June, 461.

⁴ Haupt, 'Med. Ztg. Russl.,' 1845, 375.

⁵ Bogorodsky, ib., 1854, 10.

⁶ Wilson, 'Med. Notes on China,' Lond., 1846, 22.

⁷ Friedel, 'Beitr. zur Kenntniss des Klimas und der Krankh. Ost-Asiens,' Berl., 1863, ii, 21; Albrecht, 'Petersb. med. Zeitschr.,' 1862, iii, 51; Vidal, 'L'ascaride lombricoïde en Chine et au Japon,' Montp., 1865.

⁸ Heymann, 'Krankh. in den Tropenlanden,' Wurzb., 1855, 222; v. Leent, 'Arch. de méd. nav.,' 1867, Oct., 250; 1872, Jan., 22; 1873, Févr., 102.

⁹ Young, 'Calcutta Med. Transact.,' 1826, ii, 334; Leslie, ib., 1833, vi, 61; Crespigny, 'Bombay Med. Transact.,' 1859, N.S., iv, 94; Day, 'Madras Quart. Journ. of Med. Sc.,' 1862, Jan., 23; Cleveland, ib., 1863, Jan., 26; Milroy, 'Transact. of the Epidemiol. Soc.,' 1866, ii, 150; Huillet, 'Arch. de méd. nav.,' 1868, Jan., 26.

¹⁰ Palgrave, in 'l'Union méd.,' 1866, Nr. 20, 308.

¹¹ 'Wochenbl. zur Zeitschr. der Gesellsch. der Wiener Aerzte,' 1857, 756.

groups of *Oceania* ; while the itch is quite general among the Kanakas of the *Hawaiian Islands*,¹ it is the unanimous opinion of several French practitioners² that it rarely occurs on the *Society Islands* (Tahiti), and according to Turner³ it is rare also in the *Samoa* group. From *Africa* there are accounts of its endemic prevalence on the *Mozambique*⁴ Coast, in *Abyssinia*,⁵ *Egypt*,⁶ *Tunis*,⁷ among the native population of *Algiers*,⁸ especially the Kabyles, who are eaten up by the itch ("dévorés"), as Challan puts it ; also on the *West Coast*,⁹ where the malady, known under the name of "craw-craw,"¹⁰ or by the Portuguese term "sarna," has attacked the greater part of the negro population ; and in the *Canary Islands*,¹¹ especially Gomera and Palma, where nearly the whole inhabitants are likewise permanently subject to it. In the *Western Hemisphere* the two chief regions of itch, so far as I can learn, are *Brazil*¹² and *Peru*.¹³ In Guiana it is known under the name of "maracane," in Brazil as "sarna" (the Portuguese term, but various chronic skin diseases are included under it), and in Peru as "carracha," a distinction being drawn in that country between a milder vesicular

¹ Gulick, 'New York Journ. of Med.,' 1855, March ; Duploux, 'Arch. de méd. nav.,' 1864, Dec., 486.

² Ref. ib., 1865, Oct., 291.

³ 'Glasgow Med. Journ.,' 1870, Aug., 502.

⁴ Lichtenstein, in 'Hufeland's Journ. der Heilkde.,' 1804, xix, H. 1, 180.

⁵ Ccurbon, 'Observ. topogr. et méd., &c.,' Par., 1861, 33.

⁶ Pruner, 'Krankh. des Orients,' Erlang., 1846, 142 ; Fox, 'Med. Times and Gaz.,' 1867, Feb., 165.

⁷ Ferrini, 'Saggio sul clima . . di Tunisi, &c.,' Milano, 1860, 182.

⁸ Gaudineau, 'Mém. de méd. milit.,' 1842, lii, 208 ; Deleau, ib., 230 ; Armand, 'Méd. et hyg. des pays chauds, &c.,' Par. (1853), 419 ; Bazille, 'Gaz. méd. de l'Algérie,' 1868, 39 ; Challan, ib., 117. I may remark here that the so-called "Gale bedouine" is not the itch, but Lichen simplex (see Armand, l. c.).

⁹ Boyle, 'Account of the Western Coast of Africa,' Lond., 1831, 391 ; Oldfield, 'Lond. Med. and Surg. Journ.,' 1835, Nov., 403 ; Bryson, 'Report on the Climate and Diseases of the African Station,' Lond., 1847, 258 ; Duncan, 'Travels in Western Africa,' Lond., 1847, i, 32 ; Daniell, 'Sketch of the Med. Topogr. of the Gulf of Guinea,' Lond., 1849, 114 ; Clarke, 'Transact. of the Epidemiol. Soc.,' 1860, i, 104 ; Abelin, 'Étude sur le Gabon,' Par., 1872, 31 ; Férís, 'Arch. de méd. nav.,' 1879, Mai, 330.

¹⁰ See O'Neill's remarks on *Filaria sanguinis* given at p. 333.

¹¹ Ref. in 'Arch. de méd. nav.,' 1867, April, 253.

¹² Sigaud, 'Du climat et des malad. du Brésil,' Par., 1844, 397.

¹³ Smith, in 'Edinb. Med. and Surg. Journ.,' 1840, April, 339.

form ("carracha fina") on the coast, and a more severe pustular form ("carracha sierrana") among the mountains.

§ 126. WANT OF CLEANLINESS THE SINGLE FAVOURING
CONDITION FOR THE ITCH.

The great prevalence of itch *in high and low latitudes* contrasting with its comparatively rare occurrence in the temperate zone, is not explained by the *circumstances of climate* any more than its common occurrence among the natives of these regions, and its small number of victims among foreigners (Europeans), is explicable on the ground of *racial or national peculiarities*. The single determining factor is the development of the sense for cleanliness corresponding to the degree of cultivation in general, the appreciation of the care of the skin, and of its careful treatment when out of order; in short, the degree of consideration given by the individual or the community to the withstanding of injurious influences (among the rest the itch insect) and to the combatting of the same. Not only is the number of cases dependent thereon, but also the intensity which the disease attains to; for where the itch is most widely diffused, there also it induces the severest forms of skin disease,—the pustular, impetiginous, and ecchymatous eruptions, and the ulcerations that have been found among the natives of India (the most notorious being the "Malabar itch"), of Egypt, the West Coast of Africa,[†] Mozambique, Peru, and other countries.

*Sandflea*¹ (*Pulex*, *Rhynchoprion penetrans*).

§ 127. GEOGRAPHICAL DISTRIBUTION OF SANDFLEA.

This parasite is indigenous only to the tropical regions of the Western Hemisphere, from 23° N. (Mexico) to 28° S.² Quite recently (1872) it was imported³ by a vessel from Brazil into the *Gaboon Coast* and the *Congo Coast*, where it soon became a widely diffused and frightful scourge to the natives.⁴ In the Western Hemisphere, where the first European immigrants soon made the acquaintance of this extremely troublesome insect, its range includes *Mexico*⁵ (both the eastern and western provinces), all the States of *Central America* both on the Pacific and Atlantic side,

¹ The colloquial names for this parasite are chigue, chigo, or tchike in the West Indies, sikka in Guiana, bicho, tunga, jatecuba, and migor in Brazil, nigua in Mexico, and picque in Paraguay.

² It is only the female that can be considered a true parasite, and she only for the reproductive period, or up to the maturation and extrusion of the eggs. The animal bores through the human skin with its head (particularly under the toe-nails), while its body remains free above the skin. After the eggs have been extruded, the animal dies, shrivels up, and drops off from the skin of its host. The unimpregnated females and the males feed on blood from time to time, like *Pulex irritans*. See Karsten, 'Beitr. zur Kenntniss des *Rhynchoprion penetrans*,' Mosk., 1864, p. 59; Guyon, 'Gaz. méd. de Paris,' 1863, p. 163; 'Compt. rend.,' 1870, lxx, 785.

³ Falkenstein, in 'Virchow's Arch.,' 1877, vol. lxxi, p. 436; Ballay, 'L'Ogooue,' Paris, 1880, p. 41; Rey, 'Annal. d'hyg.,' 1880, Juin, p. 496.

⁴ According to Skripitzin, a Russian physician (quoted in the 'Zeitschr. für die ges. Med.,' 1840, xiii, 76, from the 'Gesundheitsfreund' (1838), a Russian journal of popular medicine), *Pulex penetrans* would have been domesticated in Africa before the date in the text, inasmuch as it was found upon negroes during their shipment from the Congo coast or Mozambique to America. This statement stands quite by itself, and I believe it to be a mistake, as the medical accounts from the Mozambique coast make no reference whatever to the parasite.

⁵ The earliest information of it in that country was given by Chappe d'Anteroche ('Voyage en Californie,' Paris, 1772, p. 20). Later accounts will be found in Uslar, 'Preuss. med. Vereins-Ztg.,' 1843, Nr. 36 (for Oajaca); Vizey, 'Mém. de méd. milit.,' 1863, Octbr., 316 (for the east coast and the highlands); Lucas, 'La frégate "la Victoire" à Guaymas et à Mazatlan,' Par., 1868, 41 (for Mazatlan, on the west coast).

(Honduras,¹ Guatemala,² Costa Rica,³ San Salvador,⁴ and Panama),⁵ *New Granada*,⁶ *Guiana*,⁷ the *West Indies*,⁸ *Brazil*,⁹ and the tropical parts of *Paraguay*,¹⁰ *Chili*,¹¹ and *Peru*.¹²

§ 128. INSTANCES OF THE SANDFLEA IN EUROPE.

It thus appears that *Pulex penetrans*, as a pertinacious form of parasitic life, is strictly confined to the tropics, within which it is capable of transference from one place to

¹ Hamilton, 'Annals of Milit. Surgery,' Lond., 1864, 142.

² Bernoulli, 'Schweiz. med. Zeitschr.,' 1864, iii, 100.

³ Schwalbe, 'Arch. für klin. Med.,' 1875, xv, 347.

⁴ Guzman, 'Essai de topogr. phys. et méd. de la république du Salvador,' Par., 1869, 125.

⁵ Roulin ('Compt. rend.,' 1870, lxx, 792) publishes a letter by a Bishop of Panama, dated 1688, in which it is stated that as early as 1538 a division of Spanish troops was disabled from marching by swarms of the parasite settling in their feet. In recent times the French troops under Bazaine, in the Mexican expedition, had the same unfortunate experience.

⁶ Humboldt, 'Voyage,' vii, 250; Goudot, quoted by Bonnet, 'Arch. de méd. nav.,' 1867, Août, 95.

⁷ Bajon, 'Nachrichten zur Geschichte . . von Cayenne,' from the French. Erf., 1780, i, 91; Rodschied, 'Bemerk. über das Klima . . von Rio Esequibo,' Frankf., 1796, 306; Campet, 'Traité prat. des malad. graves des pays chauds,' Par., 1802, 454; Ferg, 'Jahrb. der deutsch. Med.,' 1813, i, 149; Nieger, 'De la puce pénétrante des pays chauds,' Strasb., 1859; Pop, 'Nederl. Tijdschr. voor Geneesk.,' 1859, iii, 213; v. Hasselt, ib., 1860, iv, 727; Bonnet, l. c., 1867, Juill, 19, Août, 81, Octbr., 258.

⁸ The earliest account of the parasite in the West Indies is given by Oviedo ('Cronica de las Indias,' 1547, fol. xxi). Subsequent notices of it occur in Sloane, 'Diseases of Jamaica,' Germ. ed., Augsb., 1784, p. 87; Moseley, 'Treatise on Tropical Diseases, &c.,' Lond., 1787; Savarésy, 'De la fièvre jaune,' Napl., 1809, 93; Labat, 'Annal. de la méd. physiol.,' 1833, Avril; Levacher, 'Guide méd. des Antilles,' Paris, 1840, p. 325; Brassac, 'Arch. de méd. nav.,' 1865, Dec., p. 510; Moulin, 'Pathol. de la race negre,' Paris, 1866, p. 26.

⁹ The first mention of it in Brazil is by Piso ('Hist. rerum natural. Brasiliens,' 1648, p. 249). Later authorities are Lallemand, in 'Schmidt's Jahrb. der Med.,' 1842, xxxv, 171; Burmeister, 'Reise in Brasilien,' 1853; Canoville, 'Des lésions produites par la chique, &c.,' Par., 1880.

¹⁰ Munck and Rosenskjøld, 'Vetensk. Akad. Förhndl.,' 1849, Nr. 2; Mantegazza, 'Lettere med. sulla America meridion.,' Milano, 1860, i, 284.

¹¹ Molina ('Saggio sulla storia naturale de Chili,' 1782, p. 214) expressly mentions that the parasite is confined to certain parts of the country, notably Coquimbo, in 29° N.

¹² Tschudi, 'Oest. med. Wochenschr.,' 1846, p. 472.

another, as shown by its introduction into the West Coast of Africa. That it can subsist for a brief period in temperate latitudes also, is proved beyond doubt by several observed facts.

An interesting case of the kind is given by Bonnet:¹ On board a hospital ship which had sailed from Cayenne (Isles du Salut) in July, 1866, one of the engineers was laid up, about the latitude of the Azores, with an abscess over the great toe, which was found on examination to be due to a sandflea that had bored its way into the tissues. On September 1st, the vessel arrived at Toulon, where she underwent repairs, and in January following she was again commissioned. A sailor who had been occupied about the bunkers while the coals were being taken in, was attacked with an inflammation of the foot between the toes, and the ship's surgeon detected at the spot a living female sandflea full of eggs. It follows that the insect had survived more than six months, under very various conditions of weather and climate. A similar case came under Laboulbène's² observation in a man who had arrived in Paris from Pernambuco, two living specimens of sandflea full of eggs having been found in the skin of the little toe.

§ 129. SANDFLEA ACQUIRED IN FILTHY HUTS AND PIGGERIES IN THE TROPICS.

Generally speaking, *Pulex penetrans* occurs more frequently on low plains, especially the *sea coast* or the *banks of rivers*, than on high ground in the interior;³ although the observations of Uslar and Vizy in Mexico show that it has been met with also at considerable elevations. The headquarters of the parasite are everywhere filthy and dilapidated human dwellings such as the huts of negroes and Indians, or cattle sheds, and most of all piggeries. It occurs also in houses that are kept with cleanliness, but very rarely,⁴ and under the circumstances stated in § 130. Not a single case of pulex-invasion was found by Vizy among the French troops quartered in the town of Orizaba or in the monasteries which had been converted into barracks; whereas many cases occurred among those sections of the troops who

¹ L. c., p. 99.

² 'Annal de la soc. entomol. de France,' 1867, sér. iv, tome vii, Bull., p. 6.

³ Brassac, Bonnet.

⁴ Karsten, Vizy, Bonnet, Carnoville.

lived in the half-dilapidated Indian huts, as well as among the Mexican troops located in the suburbs where herds of swine were roaming about the adjoining gardens, and among a division of Zouaves who lived in sheds that had formerly been used for keeping pigs in.

§ 130. SANDFLEA ENTERS HOUSES IN THE RAINY SEASON.

A very *high temperature* and very copious *rainfall* are destructive of the insect as well as of its eggs and larvæ. Accordingly when that kind of weather sets in, the larvæ may be seen to bury themselves in sand or rubbish-heaps. Meanwhile the mature insects take refuge in the darkest corners; they resort at such times even to dwelling houses, where they take up their quarters among the ashes or wood-shavings, or in the dustbin or rubbish-heap, or in the unboarded floors of rooms. In this way we explain the fact of the inhabitants suffering most from the parasite at the end of the dry season and the beginning of the rains.

§ 131. PERSONS MOST LIABLE TO SANDFLEA.

Race or *nationality* have no influence as regards immunity from or invasion by the parasite. Indeed, newly-arrived strangers suffer on the whole more frequently and more severely than the natives or the acclimatised, the explanation being, as Harsten¹ points out, that "strangers pay no heed to the faint itching which the animal causes in boring through the skin, not knowing the significance of that slight pain; and the animal once in its nest gives no further trouble, if the slightly inflamed bit of skin where it is located be not pressed upon or scratched." Bonnet's² view is the same; and he points out that the Indian tribes in Cayenne protect themselves against the sandflea by anointing the skin with "orleans" (roucou), and that the women always carry one or two needles with which to remove the insect the moment it has got into the skin. Speaking of the coolies from India

¹ L. c., p. 58.

² L. c., p. 101.

who come to Cayenne to work on the French plantations, Bonnet says, “ Ils sont, à leur arrivée, littéralement dévorés par les puces pénétrantes. Un de ces hommes provenant des mines aurifères de l’Appronage, avait plus de 300 sacs de chiques disséminées dans diverses régions du corps.”

As regards the individual his chance of protection against this parasite, as against others, will depend on the degree of cleanliness which he practises, and on his carefulness not to expose the naked feet in those places where the insect lurks, inasmuch as the feet are the parts that it mostly attacks. It is generally found on the toes, particularly under the nails, being rarer in other parts of the foot, and only now and then met with in the skin of the scrotum, knee, upper extremity, neck and back.

Diptera.

§ 132. LARVÆ OF FLIES IN THE NASAL PASSAGES.

An extremely painful and not unfrequently dangerous affection of the *nasal passages* is that which is caused by the entrance into them of the *larvæ of flies*. The danger, when it occurs, is dependent on the fact that the inflammatory process produced by the larvæ undergoing their development, does not remain confined to the mucous membrane of the nasal cavity, but extends to that of the frontal sinuses and the antrum, where it causes an ulcerative process and ultimately caries, and sometimes even perforation into the cranial cavity and fatal meningitis. In other cases the larvæ have found their way into the throat and even into the larynx, where they have been no less destructive in their operations.

This disease has been observed at many parts of the world in all latitudes; but it has been especially frequent in several regions of the tropics, and in these it has to some extent the character of an endemic. This applies particularly to *India* (where the disease is known under the colloquial name of

“peenash”),¹ *Mexico*,² *Central America*,³ *Cayenne*,⁴ *New Granada*,⁵ *Brazil*,⁶ and the *Argentine Republic*,⁷ where the malady bears the name of “myiasis” or popularly “bicheiro;” also to *Senegambia*, where, according to Béranger Féraud,⁸ all the cases observed hitherto have come from one locality, the district of Thiès lying to the south-east of Cayor, although it is probable that the disease is indigenous to all the lower part of the Colony.

§ 133. LARVÆ FOUND IN THE NASAL PASSAGES BELONG MOSTLY TO TROPICAL SPECIES.

This preponderance of the disease in the tropics is doubtless to be explained by the circumstance that it is only larvæ of certain species of flies proper to tropical regions which make the nasal cavities of man their favourite resort. Even if we admit the fact put forward by Frantzius, Moore, Brandão and others, that it is those persons suffering from ozæna who are mostly affected by the disease, the insect finding itself attracted to the nose by the smell coming from it—even if this should pass unchallenged, it is still obviously insufficient to explain the comparative frequency of the malady in the tropics, inasmuch as there is no evidence

¹ McGregor, ‘*Calcutta Med. Transact.*,’ 1829, iv, 28; Lahory, ‘*Ind. Annals of Med. Sc.*,’ 1855, Oct.; Rustomjee, ‘*Bombay Med. Transact.*,’ 1861, N.S., vi, App. xxviii, 1862, N.S., vii, App. xxi; Day, ‘*Madras Quart. Journ. of Med. Sc.*,’ 1862, Jan., 37; Lyons, ‘*Indian Annals of Med. Sc.*,’ 1862, May, 55; Moore, ‘*Med. Times and Gaz.*,’ 1869, Aug., 269.

² Weber, ‘*Mém. de méd. milit.*,’ 1867, Févr., 158; Lucas, ‘*La fregatte “la Victoire” à Guaymas et à Mazatlan*,’ Par., 1868, 47.

³ Frantzius, in ‘*Virchow’s Arch.*,’ 1868, xliii, 98; Bernoulli, ‘*Schweiz. med. Zeitschr.*,’ 1862, iii, 100.

⁴ Coquerel, ‘*Arch. gén. de méd.*,’ 1858, Mai, and ‘*Compt. rend. de la soc. de biologie*,’ in ‘*Gaz. méd. de Paris*,’ 1858, 430; Daunt, ‘*Dublin Med. Press*,’ 1860, Sept.; Audonit, ‘*Des désordres produits chez l’homme par les larves de la Lucilia hominivorax*,’ Par., 1864; Gourrier, ‘*Arch. de méd. nav.*,’ 1879, Juin, 471; Prima, ‘*Considérations sur la Lucilia hominivorax*,’ Par., 1881.

⁵ Saffrey, ‘*Tour du monde*,’ 1873, Sem. ii, 100.

⁶ Daunt, l. c.; Brandão, ‘*Revist. med. da Bahia*, 1876, Dec., in ‘*Arch. de méd. nav.*,’ 1877, Avril, 314.

⁷ Conil, ‘*Act. de la Acad. nacional de ciencias*,’ 1879, iii, 69, and ‘*Nouv. cas. de myiasis observés dans la province de Cordova*,’ Cord., 1880.

⁸ ‘*Maladies des Européens au Sénégal*,’ Par., 1875, i, 233.

that an amount of ozæna proportionate to the parasitic affection occurs there any more than it does in other latitudes.

It is probable that the insects which mostly give rise to the malady are various *species of Calliphoræ*; but the chief part would appear to be played by a species described by Coquerel under the name of *Lucilia hominivorax*, being perhaps identical with *Calliphora vomitoria*, or the "varejeira" (Brandão), and with *Calliphora anthropophaga* (Conil).

According to several observers the principal occasions of the insect entering the nose are when the individual is in a state of drunken unconsciousness or sound asleep in the open air.

§ 134. BOTS IN THE HUMAN SUBJECT.

Another diptera-disease of man, observed, like the former, unusually often in particular parts of the world, is the so-called "*bots*," or the *larvæ of various species of Estrus developing in the subcutaneous connective tissue*. The first information of a quasi-endemic prevalence of this affection came from Cayenne, where the parasite is known under the name of "*ver macaque*"¹; there are accounts of it also from *New Granada*² and *Brazil*,³ and, according to Frantzius, it is somewhat common in all the warmer and moister parts of *Central and South America*.

Belonging to the same group, possibly, is a disease known in *Peru* under the name of "*uta*," which is endemic, according to Smith⁴ and Tschudi,⁵ in various parts of that country, particularly in the Quebrada of Santa Rosa de Quibe, on the road from Lima to the mines of Cerro Pasco; it consists of an inflammation caused by the boring of a parasite through the skin, generally of the scrotum, the sequel being an ulcer which takes on a cancerous (?) or lupous (?) character, and ends fatally in unbearable pain and severe hæmorrhages.

¹ Thion de la Chaume in his translation of Lind, 'Essai, &c.,' Par., 1785, 75; Bonnet, 'Du parasitisme,' Montp., 1870.

² Goudot, 'Annal. des sc. nat.,' iii, 221.

³ Account in 'Gaz. med. da Bahia,' quoted by Rey, 'Annal. d'hyg.,' 1880, Juin, 501.

Edinb. Med. and Surg. Journ., 1840, April, 339.

Oester. med. Wochenschr., 1846, 509.

From high latitudes also, there are accounts of similar endemics of bots, such as the one given by Spence¹ for the *Shetland Islands*, and those by Höegk,² Thesen,³ and Böeck⁴ for various parts of *Norway*. For obvious reasons the malady is found oftener among country people who are occupied with cattle-tending and who sleep out of doors. It is a noteworthy fact that the parasite in man is nearly always on the head or trunk, and only exceptionally on the extremities.

¹ 'Edinb. Med. Journ.,' 1858, Nov., p. 417.

² 'Norsk Magaz. for Lægevidensk,' 1869, xxiii, 489.

³ *Ib.*, 1872; 'Nord. med. Selsk. Fordhl.,' 89.

⁴ *Ib.*, 1871; 'Selsk. Fordhl.,' 227.

CHAPTER X.

PARASITIC FUNGI.

§ 135. PITYRIASIS VERSICOLOR AND FAVUS.

Of the parasitic fungi we have to consider here only those which produce diseases of the skin, the so-called dermatomycoses. Two of these, pityriasis versicolor and favus, have little of interest for geographical inquiry. Both diseases, or their respective fungi,—the *Microsporon furfur* and *Achorion Schoenleinii*—would appear to be distributed universally over the globe, without attaining a properly endemic character at any one place.

The comparatively large amount of favus among the Jewish population, particularly the male part of it, in *Russia*, *Poland*, *Hungary*, *Galizia*, and the *Levant*,¹ as well as among the Mohammedan population in *Turkey*,² *Asia Minor*, *Syria*,³ *Persia*,⁴ *Egypt*,⁵ *Algiers*,⁶ and *Morocco*,⁷ is perhaps to be explained by their religious practice of always going with the head covered. It is not so easy to account for the frequent occurrence of the malady in *France* (particularly the southern provinces),⁸ and in some parts of *Italy* (again

¹ Eder, 'Zeitschr. der Wien. Aerzte,' 1853, i, 244; Zeissl, 'Oest. Zeitschr. für pract. Heilkde.,' 1864, Nr. 31.

² Rigler, 'Die Türkei und deren Bewohner,' ii, 80.

³ Pruner, 'Krankheiten des Orients,' 149.

⁴ Polak, 'Wochenbl. zur Zeitschr. der Wiener Aerzte,' 1857, 743.

⁵ In the military hospitals there is a special department for favus patients. See Fox, 'Med. Times and Gaz.,' 1867, Feb., p. 165.

⁶ Armand, 'Méd. et hyg. des pays chauds, &c.,' 419; Challan, 'Gaz. méd. de l'Algérie,' 1868, 119; "la teigne se rencontre à chaque pas," he says.

⁷ Account in 'Med. Times and Gaz.,' 1877, July, 97.

⁸ Bergeron, 'Bull. de l'Acad. de méd.,' xxx, 1864, 20, 27, Dec. He estimates the number of favus patients in the Herault district at 20 per 1000 of the population.

mostly in the southern provinces,¹ such as Apulia, Capitanata, the Abruzzi, Basilicata and Calabria); nor the fact, often adverted to, that favus is much commoner in *Scotland*² than in England, being met with, as in France, to a much greater extent among the country people than among the residents in towns, although the case is exactly the opposite with herpes tonsurans; nor, finally, to explain why favus is quite unknown in the Island of Martinique, as Ruz³ and Bergeron⁴ agree in saying that it is.

There can be no question here of influences of *climate*⁵ and other general causes, or of a physiological predisposition in the individual towards or against, depending on circumstances of *race* and *nationality*. We are in fact driven to believe that the occurrence of Achorion Schoenleinii is associated with certain conditions of locality, its transmission taking place more readily under some circumstances than under others; and the determining factor is possibly the greater or less amount of favus among the domestic animals, from which it is well known to be often transmitted to man. As we know nothing of the existence of the fungus away from the body of man or other animals, it is impossible for the present to form any definite opinion as to what those circumstances are. What we may conclude is that *want of cleanliness* is one of the chief factors in the diffusion of favus and pityriasis versicolor, as of all other communicable dermatomycoses.

¹ Lombroso, 'Rivista clin. di Bologna,' 1872, 225; Sormani, 'Geogr. nosol. d'Italia,' Roma, 1881, 321. From the conscription lists for the years 1864-77, the number of favus patients in Italy is estimated at 9·1 per 1000 inhabitants; but while the proportion in the southern provinces named in the text is 20 per 1000 and upwards, it is only 2 per 1000 in the northern districts of Casale, Alba, Cuneo, Belluno, Padua, Rovigo, Florence, &c. The number of cases has decreased considerably of late.

² Anderson, 'Lancet,' 1871, Nov., 743; Bennett, 'Edinb. Monthl. Journ. of Med. Sc.,' 1850, July, 40.

³ 'Gaz. méd. de Paris,' 1859, 419.

⁴ 'Annal. d'hyg.,' 1865, xxiii.

The fact that favus is often seen in high latitudes, as in Kamschatka (Bogonodsky, 'Med. Ztg. Russlands,' 1854, No. 1), shows that it is not the warm climate of Southern Europe and the East which determines its occurrence.

§ 136. HERPES TONSURANS OR RINGWORM.

Ringworm plays a considerably greater part than favus or pityriasis in the statistics of sickness, especially from tropical countries, in many of which this parasitic skin disease has a truly endemic character. We have more particular accounts of it from *India*,¹ *the Malay Archipelago*,² *Cochin China*,³ *China*, *Japan*, several of the island-groups of *Oceania* (of which more in the sequel), *Nossi-Bé*⁴ near Madagascar, *Abyssinia*,⁵ *Egypt*,⁶ *the West Coast of Africa*,⁷ *the West Indies*,⁸ *Guiana*,⁹ and *Peru*.¹⁰ Even in the *higher latitudes of the Eastern Hemisphere*, herpes tonsurans is much more common than favus; and the same is true of the *North American continent*.¹¹

The preponderance of the disease in the tropics warrants the conjecture that the *climate*, or a *high temperature* and a large amount of *moisture*, are a material furtherance to the *Trichophyton tonsurans*; and this idea finds support in the fact that the amount and severity of the malady increase in the rainy and hot seasons, while they diminish in the cold season.¹² Leclerc states that patients with ringworm coming

¹ Voigt, 'Bibl. for Laeger,' 1833, July, 2; Young, 'Calcutta Med. Transact.,' 1826, ii, 334.

² Lesson, 'Voyage méd. autour du monde,' Par., 1829, 98; Heymann, 'Darstell. der Krankh. in den Tropenländern,' Würzb., 1855, 202; v. Leent, 'Arch. de méd. nav.,' 1867, Octbr., 250; 1873, Févr., 102.

³ Leclerc, 'L'herpes circiné en Cochinchine,' Montp., 1871; Beaufils, 'Arch. de méd. nav.,' 1882, Avril, 276.

⁴ Corre, ib., 1878, Novbr., 408.

⁵ Blanc, 'Gaz. hebdom. de méd.,' 1874, Nr. 21, 330.

⁶ Pruner, 'Krankh. des Orients,' 149; Hartmann, l. c.

⁷ Thaly, 'Arch. de méd. nav.,' 1867, Sptbr., 187; Abelin, 'Étude sur le Gabon,' Par., 1872, 31.

⁸ Hillary, 'Diseases of Barbadoes,' Germ. ed., Leipz., 1776, 420; St. Vel, 'Malad. des régions intertropicales,' Par., 1868, 484.

⁹ Nissaeus, 'De nonnullis in Colonia Surinamensi observat. morb.,' Harderovici, 1791; Rodschied, 'Bemerk. über das Klima . . von Rio Essequibo,' Frankf. a. M., 1796, 256.

¹⁰ Smith, 'Edinb. Med. and Surg. Journ.,' 1840, April, 339.

¹¹ Bulkley, 'Chicago Med. Journ.,' 1877, Nov.; 'Transact. of the American Dermatol. Soc.,' 1878, 28.

¹² Fox and Farquhar, 'On certain Endemic Skin and other Diseases of India, &c.,' Lond., 1876; Leclerc, l. c.

from Cochin China to Europe improve considerably as they enter the higher latitudes, but a fresh access of the disease occurs when they approach the tropical heat of the Arabian coast. The fact of ringworm occurring chiefly among the coloured population of the tropics, which has been dwelt upon by a number of observers such as Pruner for the negroes in Egypt, Leeson for the Malays of the East Indies, van Leent¹ for the Chinese in Banka, and Corre for the Malagasys in Nossi-Bé, is to be explained not so much by *racial differences* as by *the circumstances of living*, that is to say, by want of cleanliness and by carelessness, which give considerable facilities for the spread of the parasite and may even raise the malady to an epidemic.² It is an established fact that the parasite is often transmitted to man by domesticated animals such as the cat and dog.

Various forms of Herpes tonsurans.—Herpes tonsurans, as is well known, occurs in a variety of forms, which have given rise to various synonyms of the malady, such as porrigo scutulata, herpes circinatus s. annularis (ringworm), sycosis parasitaria, and onychomycosis. The following paragraphs relate to skin diseases concerning which it is impossible to decide with certainty whether they are merely varieties of herpes tonsurans, depending on climatic or other circumstances, or whether they are peculiar species of dermatomycoses.

§ 137. GUNE,³ TOKELAU RINGWORM (LAFA TOKELAU), AND CASCADÖE.

Under the name of "gune," Fox⁴ gave the following description of a skin disease which was prevalent in endemic form in the *Kingsmill* or *Gilbert Group* (under the line in

¹ 'Arch. de méd. nav.,' 1873, l. c.

² Gorley ('Lyon méd.,' 1880, No. 27, p. 28) gives an account of an epidemic of *Trichophyton tonsurans* at Fernay. It began in a barber's shop, to which the parasite had been brought by country people, and it spread through a large circle of the inhabitants.

³ The term "gune" means, not skin disease, but "skin" (Königer, in 'Virchow's Archiv,' 1878, vol. lxxii, p. 414).

⁴ In Wilkes's 'Narrative of the U.S. Exploring Expedition, 1844,' vol. v, p. 104.

172 E.), and in a few of the neighbouring island-groups of *Oceania* :

This disease, which closely resembles ringworm in the stages of its development, begins as a small ring covered with scales, about an inch in diameter; this circle increases gradually, another ring forms inside it, and a new ring inside that, which has meanwhile enlarged considerably. It often happens that a number of rings arise in one part of the body side by side, which coalesce and give rise to various forms of serpiginous and concentric figures. In the end the whole body appears to be covered with a scaly deposit which always produces very intense itching. When the scales fall off, there remain behind innumerable rings and sinuous lines of a livid colour, which disfigure the individual very much, and not unfrequently for the whole of his life, although his general health does not appear to be affected.

Tokelau or Union Island Ringworm.—According to Turner,¹ who gave the disease the name of herpes desquamans to distinguish it from the ordinary herpes tonsurans, this malady was introduced from the Gilbert Islands into the *Union* or *Tokelau Group* (particularly into Bowditch Island) and subsequently into the *Samoa Islands*. From the former of these it got the name of *Tokelau ringworm* (Lafa Tokelau). Königer,² who has had opportunities of studying the disease in the Samoa Islands, says that the importation from Tamana (Gilbert Islands) had probably not taken place before 1860, that it had attained only a limited diffusion in Samoa at the time of his residence there (1872), and that it had shown itself in the Caroline and Pelew Islands as well. His account is as follows :

The malady begins with an eruption of small papules mostly grouped in circles, which cause intense itching and desquamation of the epidermis around their growing periphery. Afterwards these circular efflorescences coalesce, the skin becoming at the same time hard, dry and brittle, and, in the end, when the disease has become diffused over the whole body, the skin looks as if covered with a clayey substance. An examination shows that this substance consists of epidermic scales of various sizes up to half an inch, which are in part detached and in part still adhering to the skin. The hair of the body is almost gone at the places where the eruption occurs, but not the hair of the head, where the eruption is very scanty, as it is also on the face. The only inconvenience to the patient is the itching, which is often unbearable.

¹ 'Glasgow Med. Journ.,' 1870, Aug., 502.

² 'Virchow's Arch.,' 1878, Bd. 72, 413.

Microscopic examination shows that the seat of the malady is limited to the epidermic layers. Among the epidermic cells there are more or less dense strata of mycelial threads with transparent colourless contents, as well as spherical cells filled with homogeneous substance occurring either in groups or singly. Königer has found the mycelium only in recent cases.

Macgregor¹ has lately met with the same disease in the *Fiji Islands*, but only in persons who had come from the *Solomon Islands*, the *New Hebrides* or the *Lime Islands*, never in natives of Fiji or in Europeans. His account of the skin affection agrees almost exactly with that of Königer, including the statement that the hairy scalp and the face and forehead are almost never attacked.

Further accounts of the same peculiar dermatomycosis (*i. e.* a skin disease identical with Tokelau ringworm) have been given by Corre² for the Island of *Nossi-Bé*, and by Manson³ for *Malacca* and the *Malay Archipelago*. Corre found it among the Malagasys and Anjouanais; Manson (who speaks of it under the name of "tinea imbricata") met with a few cases of it also in China and other parts of Eastern Asia, but only in individuals who had come from Malacca or elsewhere in the East Indies.

Cascadoe.—We may perhaps include in this group of parasitic skin diseases the malady described by Pompe von Meerdervoort,⁴ under the name of "cascadoe," which he found in 1859 to be endemic to the extent of 5 per cent. of the population in several of the *Moluccas* (Gisser, Calietaroe, Ceram, Ceram Laut, Goram, and the Aroe Islands).

This malady, which always begins to develop at the earliest periods of childhood (second to fourth year), is characterised in the first instance by an outbreak of round or oval spots of a grey colour, either in perfectly symmetrical rings or scattered about irregularly; they occur first on the back and chest, afterwards on the extremities, and are soon followed by more or less troublesome itching accompanied by peeling of the skin. At a later stage, the skin is found to be dry, hard and parchment-like, the dark-coloured spots become covered with fine scales, and the condition extends gradually and uniformly over all the skin,

¹ 'Glasgow Med. Journ.,' 1876, July, 343.

² 'Arch. de méd. nav.,' 1878, Novbr., 408.

³ 'Med. Reports of the Imperial Maritime Customs for China, 1879,' abstract in 'Med. Times and Gaz.,' 1879, Sept., 342.

⁴ 'Nederl. Tijdschr. voor Geneesk,' 1859, iii, 629.

excepting that of the face which always remains free. At a still later period the scales are larger, thicker, and of horny consistence, and the skin now looks as if it were coated with a layer of clay (whence the name of "huitklei" or clay-skin) which had cracked in many places, forming fissures of various depths. Apart from the itching of the skin the patient is in no way inconvenienced.

§ 138. RELATION OF THESE LOCAL VARIETIES OF RINGWORM TO HERPES TONSURANS.

Observers are not at all agreed, as we have already remarked, in their view of the relation of the above group of skin diseases to herpes tonsurans. Tilbury Fox¹ and Farquhar are in favour of an identity of origin for the various forms of disease described as "Burmese," "Indian," "Chinese" and other ringworms named after countries, assuming that the condition is in all cases due to *Trichophyton tonsurans*, and that the variety of form depends merely on the external influences. Manson and Macgregor, again, are satisfied that the parasite in Tokelau ringworm is a different one from *Trichophyton*; while Corre and Königer have not come to a decided conclusion. The arguments which have been adduced (and, it seems to me, with good reason) against the genetic identity of herpes circinatus and Tokelau ringworm are based upon the difference between the two forms as regards type of disease and natural history of the parasite.

Regarding the type of the disease, it is a noteworthy fact that Tokelau ringworm, like "cascadoe," never occurs on the hairy parts of the head—a fact which Corre would attach all the more importance to from having met with numerous cases of ordinary herpes tonsurans on the hairy scalp among children in Nossi-Bé; that it spreads, on the other hand, often over the whole body, in contrast to the habit of herpes circinatus; and finally, as Königer points out, that the spots in herpes circinatus break out with more intense inflammatory accompaniments than in Tokelau ringworm, the redness of the skin and formation of vesicles being more considerable, while, in the later stages of herpes

¹ 'On certain Endemic Skin and other Diseases of India, &c.,' Lond., 1876, 59.

circinatus but never in Tokelau ringworm, the spots heal at the centre and extend at the periphery, so as ultimately to form rings of several inches diameter.

Regarding the life-history of the parasite itself, Macgregor observes that the mycelial threads are much more numerous while the spores are fewer and smaller, in Tokelau ringworm than in circinate herpes; and Manson states that the spores in the former are of an oval form, and that the mycelial threads do not show those swellings and constrictions which have been observed in Trichophyton. Moreover, we learn from Manson that the inoculation experiments which he has frequently practised with the fungus of Tokelau ringworm, have in all cases produced that particular type of disease, and never herpes circinatus.

Mal de los pintos.

§ 139. CLINICAL CHARACTERS OF THE PINTA DISEASE.

Under the name of “mal de los pintos” or other colloquial synonym,¹ a highly remarkable skin disease has been described, which would appear to be endemic exclusively in a few tropical regions of the Western Hemisphere, particularly on the slopes of the Cordillera. The mycotic nature of this disease has been placed beyond doubt by the recent paper of Gastambide.²

¹ Pinta, mal pintado, tinna (in Mexico); cute or carate (in Venezuela and Granada); quirica (in Panama).

² I give here in alphabetical order the names of writers on the disease so far as known to me:—Alibert, in ‘Revue méd.’ 1829, Aug., p. 228 (based on a case under his own observation, and on reports from Granada by Zea, Bonpland, Daste, and Roulin); Burkhart, ‘Aufenthalt und Reise in Mexico,’ Stuttg., 1837 i, 213; Chassin (based on a memoir by Gomez communicated to the Institut de France, but not published); Gastambide, ‘Presse méd. belge,’ 1881, Nr. 33, 35, 39, 41; Girard, ‘Relation méd. de la campagne de la frégate “le d’Assas” dans les mers du Sud, &c.,’ Montp., 1868, 13; Gomez, ‘Du carathès ou tache endémique des Cordillères,’ Par., 1879; Heller, ‘Wiener Sitzungsber.,’ 1848, Nr. 3, 122; Iryz, ‘Independencia Medica,’ 1882, Jan., abstract in ‘Brit. Med. Journ.,’ 1882, Novbr., 903; McClellan, ‘Lond. Med. Repository,’ 1826, xxvi, 167; Mühlenpfordt, ‘Versuch einer Schilderung der Republik Mexico,’ Stuttg., 1844 i, 355; Müller, ‘Monatsbl. für med. Statistik,’ 1847, 43.

The disease comes out in the form of spots of various discoloration, with scaling of the epidermis over the affected parts of the skin and more or less intense itching, the general well-being of the patient remaining otherwise unaffected. According to these characters it corresponds most closely, among diseases classed as dermatomycoses, to pityriasis versicolor.

In exceptional cases the outbreak of the malady is preceded by a series of symptoms¹ of a general kind, such as chills and heats, headache, thirst, loss of appetite, sickness and it may be vomiting, diarrhœa and profuse sweating. These continue from four to seven days, after which there is a pause of some forty days, and then the first signs of the skin affection appear.² Usually, however, the disease develops quite gradually as a purely local affection, and without producing any sign whatsoever of constitutional disturbance.

As regards colour, form, size and number, the spots show great differences in different cases. According to the colour, they may be divided into black or greyish, blue, red and dull white. It often happens that all the discolorations in one person have the same character; in other cases they show various colours at different parts of the body, so that those affected present a very striking piebald appearance. In the latter cases, the spots at the beginning of the disease are usually all of one colour, and it is not until a later stage that spots of other colours begin to appear on parts of the skin hitherto unaffected.

A change from one kind of discoloration to another has never been observed, the shade of colour assumed from the outset in each spot remaining the same throughout the whole course of the disease. Sometimes the skin affection is confined to one small area of the surface of the body, at other times the spots occur in great numbers. From small beginnings they increase in size on all sides as the disease advances, and they may even become confluent; in this way they extend by degrees over a large part of the body or over the whole of it, including the hairy parts of the head; the palms of the hands and soles of the feet are the only parts where they are never seen. Usually they show themselves first on the extremities and the face, that is to say, on the uncovered parts of the body. In form they are either somewhat rounded or quite irregular, and either sharply defined or with margins obliterated, passing by gradual transition into the normal colour of the skin. Pressure on the spots makes no difference to their colour, and it may be seen at the same time that they are on a level with the skin

¹ The sketch of the disease that follows is mostly based upon the latest and most complete accounts of it by Gomez, Iryz, and Gastambide.

² Gastambide and Iryz make no reference to this prodromal stage. It appears to me to be doubtful whether there is in reality any intimate connexion between the skin disease and these symptoms, which point clearly to gastric disorder.

and not raised. When the malady has been in progress for some time, the skin at the affected places looks for the most part rough and dry; more rarely it is moist and greasy to the feel. Meanwhile, desquamation of the epidermis had begun with the first development of the affection and continues during the whole course of it, having a furfuraceous character at the outset, but with the scales several millimetres in diameter later on. When the lesion attacks the hairy parts of the body, the hairs turn white and thin, and at length fall out. Along with the desquamation, and in proportion to its copiousness, there is always a more or less troublesome amount of itching, which is for the most part acutest in the first hours of the night, so that it interferes with the patient's sleep.

Another noteworthy thing is an obnoxious smell which the patient diffuses around him, compared by some observers to the smell of foul linen that has been left in a damp place, and by others, to the odour of cat's urine. No one has ever noted symptoms of constitutional illness in this skin disease; excepting for the disagreeable itching, the general health of the patients is nowise disturbed, and they are able to go about their usual business.

The course of the disease is always chronic, months and sometimes years elapsing during which the skin affection may remain stationary with only a slight extension over the body. This applies particularly to the red and white variety, whereas in the black and blue form the diffusion over the surface is usually more rapid and more general. If the patient takes proper care of himself (above all observing strict cleanliness), and if he is properly treated, the malady will disappear, although it is easily brought back; where these conditions are not observed, it may last through the patient's whole life-time.

§ 140. HISTORY OF THE PINTA DEFECTIVE.

In the *history of the "mal pintado,"* nothing is known with certainty of the time and place of its origin. Gastambide states that it was prevalent in southern parts of America before the Spanish conquest of Mexico, and was imported into Mexico subsequently. M'Clellan assigns the earliest appearance of it in Mexico to the year 1775; it is said to have shown itself first in the northern part of the Province of Valladolid in the neighbourhood of Jurillo, and shortly after the first eruption of that volcano, and to have spread from that point southwards as far as Mascala.

§ 141. PRESENT GEOGRAPHICAL DISTRIBUTION OF THE PINTA.

The present *distribution-area of the pinta*, so far as it can be made out with certainty, includes Mexico, Central America, Venezuela, New Granada, Peru and Chili.

In *Mexico*¹ it is endemic only in the "tierra caliente" of the west coast, particularly in the Provinces of Guerréro (at Acapulco), Valladolid and Michoacan; it occurs more rarely (never according to M'Clellan) in the "tierra templada;" and its farthest range eastwards is into the western districts of the Province of Tabasco, where Heller saw it on the banks of the Grijalva. The East Coast of Mexico appears to have had no visitation of the malady. Heine-mann² expressly says that he had seen no case of it at Vera Cruz, and he seems to indicate that the discolorations of skin which he saw among mestizzos at Oaxaca had nothing in common with the mal pintado. Regarding the frequency of the disease, Gastambide says that in some villages of the above-mentioned districts of Mexico, 9 per cent. of the inhabitants are affected with it; and M'Clellan in 1826 saw at the capital a whole regiment composed exclusively of pintados.

In *Panama* it would appear from the statement of Gomez³ to be rare.⁴ In *Venezuela* the endemic seats of the malady are the Provinces of Barquisimeto and Merida.⁵ But its widest distribution is in *New Granada*; throughout almost the whole of that country it is endemic, more particularly in San José de Cucuta, in the basin of the Meta, in Cundinamarca, in the valleys of Guaduas, Tocamina and La Mesa, in the lower valleys of the Provinces of Tolima and Antioquia, in Cauca, in the districts of Valencia, Fonseca, La Paz, Soldado, and other districts on the Magdalena, and in most other parts of the Magdalena Province. There is a

¹ Müller, M'Clellan, Mühlenpfordt, Burkhart, Girard, Heller, Gastambide.

² 'Virchow's Archiv,' 1873, lviii, 189.

³ L. c., 22.

⁴ It is a question whether the "leprous spots" mentioned by Young ('Narrative of a Residence on the Mosquito Shore,' Lond., 1847, p. 26) as occurring among the natives of the Mosquito Coast were not of this nature. (See p. 28.)

⁵ Gomez, p. 21.

want of definite information about the distribution of the disease in *Peru* and *Chili*; Gastambide mentions the mal pintado as occurring in the former, and there is a brief notice of its existence in *Chili* in one of Pöppig's papers.

So far as we may conclude from what we know at present of its area of distribution, the pinta belongs to the *class of highly tropical diseases*. In Mexico it is endemic only in the "tierra caliente" of the west coast at altitudes up to 400 or 500 metres (1500 feet),¹ and in Granada only at places with a mean temperature of 20° to 30° C. (68° to 85° Fahr.).² The influence of a *high temperature* is further shown in the fact adduced by Gastambide that the recrudescence of the malady always coincides with the beginning of the warm season (spring); or, in other words, it is then that the itching of the skin becomes particularly troublesome. A *damp soil*, also, appears to help the number of cases; at least, the authorities³ are unanimous in saying that the damp or swampy banks of rivers are its chief seats.

§ 142. PREDISPOSING CAUSES.

As with all parasitic skin diseases, so with this particular form a principal factor in its existence is *hygienic neglect*, and, above all, *want of cleanliness*. It had been pointed out by M'Clellan that cases of the pinta were much rarer among the better-to-do classes in Mexico, than among the lower orders living in squalor and misery; that well-to-do people, who had among their servants persons affected with the pinta, escaped the malady, as did also the rest of the servants in the house, provided they practised the greatest cleanliness of body by means of ablutions, baths, and the like; whereas, in other cases where these precautions were neglected, the household would become infected. To the same effect, Gastambide says:⁴

"On peut observer le pinto aussi bien chez les personnes puissant d'une certaine aisance, que chez les classes pauvres. Toutefois la maladie sévit en proportion incomparablement plus forte dans les

¹ M'Clellan, Müller, Gastambide.

² Gomez.

³ Alibert, Gomez, 17, 22; Gastambide, 260.

⁴ L. c., p. 260.

classes nécessiteuses, là où les habitudes de confort et de propreté font défaut. . . . La misère avec tous ses inconvénients, le manque de propreté sont des antécédents très appropriés à l'apparition de cette affection, à son développement ultérieur, à sa durée indéfinie."

Almost in the same words, Gomez sums up his observations as to the influence of hygiene on the amount of the disease: "En résumé nous pouvons réduire toute cette série des causes à une seule—la misère." It is this then, and not any *racial peculiarity* that explains with a high degree of probability the fact of the pinta being infinitely more common among the native Indians, the negroes and the mestizos, than among the whites; and although these latter have by no means an absolute immunity from the disease,¹ yet they never take it until they have resided a considerable time at one of the disease-centres (Gastambide).

§ 143. DUE TO A FUNGUS IN THE RETE MUCOSUM.

It is only by the most recent inquiries that the true *cause of the disease* has been settled. Some authorities⁵ have adopted the notion, which used to be in general currency, that *mal de los pintos* is purely an affair of absorption of the normal pigment of the skin, or of a deposit of new pigment (in the red, blue and black varieties). Others thought that they could discover in the skin affection an indication of syphilis or leprosy; or they identified it with the dermatoses that occur in pellagra as a consequence of using damaged maize. But these opinions are contradicted by the absence of all symptoms in the pinta pointing to constitutional disturbance. Chassin laid most stress on the use of drinking-water rich in salts, especially common salt. Another belief, deeply rooted in the popular fancy, is that the skin disease follows the sting of an insect bearing the name of "jegen" or "comegen," which no one has been able to give any further account of.

Writing on the *mal de los pintos*, in the first edition of this work I expressed the opinion, that "this skin disease, like pityriasis versicolor, is an affair of an *epiphyte*;" and

¹ Alibert, M'Clellan, Heller.

² Müller, Girard.

my conjecture has lately been confirmed fully by the published observations of Gastambide :

"Microscopic examination," he says,¹ "reveals between the more deeply situated polygonal epidermic cells a deposit of small bodies, either perfectly spherical with a diameter of 8 micromill., or more oval and measuring 6 to 8 micromill. one way, and 10 to 12 the other. At the first glance they appear to be of a uniformly black colour, but when the light is made to fall obliquely they are found to be cells with their exterior formed of a transparent membrane, and filled within by a yellowish fluid having a large number of dark-coloured granular particles suspended in it, which become more obvious on adding acetic acid. Besides these cells, there are nearly always to be found fragments of tubes or tube-like filaments, which are attached to the former, according to one observer (Sandoval), like the stalk to a cherry; they are 18 to 20 micromill. broad and 2 thick, of a white colour, with a definite contour and highly refractive, showing no trace of dichotomous branching, but tapering from a somewhat broad base to a blunted point."

"La véritable cause de la maladie," says Gastambide in concluding his paper, "est donc trouvée. Il me semble incontestable que nous nous trouvons là en présence d'un parasite végétal, d'un champignon microscopique, qui par son implantation et son développement sur la peau, produit les lésions anatomiques qu'on constate dans cette affection. De ce que nous venons de dire s'en suit tout naturellement que le mal du pinto doit être rangé dans les cadres nosographiques, dans la classe des dermatomycoses."

Whether the various colours of the spots in the pinta disease are produced by various species of the fungus, or whether the differences in colour depend on the epiphyte being located in the superficial or deep layers of the epidermis, Gastambide does not attempt to decide; but the microscopic examination of the affected pieces of skin is rather in favour of the second suggestion. The black and blue spots extending in continuity over the surface depend, he tells us, upon an affection of the upper epidermic layers; they never go so deep as the rete mucosum, and when they are cured, there is no trace left of any change in the skin—which can seldom be said of the red, and still less of the white variety. These red and white spots, in fact, have their seat in the deeper layers of the epidermis. Osorio²

¹ L. c., p. 261.

² Quoted by Gomez, p. 74.

has come to the same conclusion, although he was unaware of the parasitic nature of the disease, holding it to be a simple deposition of pigment :

“La coloration du carathès,” he says, “dépend de l’arrangement et de la distribution de la matière pigmentaire, et de là des différences de nuances, qui passent du noir au bleu et du bleu au rouge, laissant des intervalles de peau sans couleur et communiquant à la peau l’aspect du marbre ou du jaspe. Quand ces intervalles sont très grands, le carathès est nommé blanc.”

§ 144. A COMMUNICABLE DISEASE.

The fact that the *mal de los pintos* is decidedly *communicable*, gives further probability to the parasitic nature of the malady. “The disease is said to be infectious,” says M’Clellan, “and facts seem to corroborate the account. I have seen persons, who were born and bred up in the higher districts, where it is not known except by report, after having lived for a few years in the low country in habits of intimacy with the people, return with the disease. Nurses who are infected with it, and have been employed in the higher districts, have communicated it to children.”

Gastambide¹ gives several typical cases of the disease being carried from place to place, and of its farther dispersion by continuous infections ; and he conjectures, as Gomez² had done, that the alleged origin of the disease from the sting of an insect, does not consist so much in the injury done, as in an infection by means of that which the insect carried.

§ 145. OTHER TROPICAL DISCOLORATIONS OF THE SKIN.

Besides these accounts of the pinta disease coming from various regions of America, and relating undoubtedly to one and the same malady, we find many other notices in the medico-topographical literature of tropical and subtropical countries referring to decolorations or discolorations of the skin, under such names as albinism, vitiligo and chloasma.

¹ L. c., p. 276.

² L. c., p. 17.

It is mostly among the coloured races that these occur; but the accounts of them are so extremely slight, so obscure, and so lacking in microscopic details of the affected parts, that it is impossible to form a trustworthy opinion as to the nature of the affections. There would be very little interest in a complete enumeration of these discolorations, mostly known to us by their vernacular names, inasmuch as nothing is known of them but the name and the colour. It is possible that many of them may be dematomycoses, some of them perhaps identical with the pinta disease itself.

For example, Sigaud¹ and Martius² give an account of cases of spotted discoloration among certain tribes of Indians in *Brazil*, the latter author writing as follows:

"The whole body seemed as if irregularly sprinkled over with blackish spots of various sizes, mostly round, and either discrete or confluent, which gave the impression of slight indurations of the skin yielding a small amount of scabby secretion, while their surface was uneven and drier than the skin elsewhere. The skin round the spots was often paler than the healthy skin, in fact, nearly white. . . . This skin disease is regarded by the neighbouring tribes as a national distinction of the *Puru-Purús*, *Amamatis* and *Catavixis*, who are on that account called 'the spotted' (*pinipinima-tapuüjo*) . . . The malady is said also to be hereditary, and even infectious."

There are other accounts from *Guiana* of a skin disease similar to the "carate;" among them a recent reference to it from *Surinam* by Pop,³ who speaks of it under the name of "lota;" and the same affection appears from the writings of Savaresy,⁴ Levacher⁵ and others to be somewhat common in the *West Indies*. Levacher's reference is to a skin disease prevalent among the negroes and mulattoes, which is characterised by numerous irregular spots of a yellowish and milky, or coffee-chocolate colour, occurring on the face, neck, chest, and other parts of the body; even in its name it would appear to have some analogy to the "lota" of *Surinam*, for the persons affected with it in the *West Indies* are known as "lotards" or "léotards."

¹ 'Du climat et des maladies du Brésil,' Par., 1844, 117.

² 'Das Naturell, die Krankheiten . . der Urbewohner Brasiliens,' München p. 66. (Reprinted from 'Buchner's Repertorium für die Pharmacie,' xxxiii, 289.)

³ 'Nederl. Tijdschr. voor Geneesk,' 1859, iii, 213.

⁴ 'De la fièvre jaune,' Napl., 1809, 81.

⁵ 'Guide méd. des Antilles,' Par., 1840, 320.

§ 146. DUBIOUS INSTANCES OF SPOTTED SKIN.

The relation to the *mal de los pintos*, and to the dermatomycoses in general, of certain discolorations of the skin in various tropical and subtropical regions of the Old World, is more questionable. Girard stands alone in thinking that he has seen the pinta disease among the negroes in *Senegambia* and the Gaboon, and I am unable to say how much weight is to be attached to his statement. We are equally at a loss to decide upon the nature of the peculiar discolorations of the skin mentioned by Pruner¹ as occurring among the coloured races in *Syria*, *Egypt*, *Arabia*, and other eastern countries; or upon the nature of a similar disease among the negroes in *Tunis* (which Ferrini² describes, moreover, as communicable), or of the variety found in *Réunion*.³ The same difficulty meets us in dealing with a skin disease found among the natives in the eastern districts of *Lower Bengal*, which consists, according to Leslie,⁴ in a peculiar spotted discoloration or decoloration of the skin, and is thought to be caused by drinking certain kinds of water.

¹ 'Die Krankh. des Orients,' Erlang., 1846, 151.

² L. c., 261. "Questo morbo e tenuto dagli indigeni in concetto di contagioso, e pare veramente che lo sia, poichè il Comm. Protomedico Lumbroso l'ha veduto diffondersi con facilità nei soldati da uno all' altro, ed anche il Cav. dott. Tagiuri vide, che se il soldato affetto da vitiligine non veniva subito riformato, egli la comunicava al vicino di letto."

³ Chapotin, 'Topogr. méd. de l'île de France,' Par., 1812, 70.

⁴ 'Calcutta Med. Transact.,' 1834, vi, 62.

CHAPTER XI.

ERYSIPELAS.

Under the title of "Infective Traumatic Diseases," we may place together three nosological forms, Erysipelas, Puerperal Fever and Hospital Gangrene, which have this much in common, that they bear the characteristics of an infective process, and are in their origin dependent on the existence of some breach of continuity in the external or internal surfaces of the body. In the three following chapters I shall inquire into the relation of those three diseases to one another and to other infective diseases, from the point of view of their history and geographical circumstances, or in their etiological aspects.

§ 147. HISTORY OF THE TERM "ERYSIPELAS." DEFINITION OF THE DISEASE.

The word "erysipelas," as the designation of an inflammatory redness of the skin, running the course of a fever and quickly extending from one point to a larger or smaller area of the surface around (hence the term "Rothlauf" in use in Germany as early as the sixteenth century), is as old as Medicine itself. In the very earliest medical writings of antiquity, the word is used in that sense; and although the opinions of the profession on the process underlying the disease, and most of all on the extent to which the term might be stretched in denoting a single morbid process, have undergone many changes, yet the name itself has continued down to the present day.

There are a few interesting references to erysipelas in the Hippocratic Collection, and more especially in certain of the books which are probably the work of Hippocrates himself.¹ He distinguishes already between idiopathic and traumatic erysipelas, or erysipelas with and without wounding; and that distinction has been maintained by the later Greek and Roman physicians,² by the Arabian³ and other practitioners of the middle ages,⁴ as well as by the whole profession in the modern period, and, to some extent, even down to the present day. Moreover, the term "erysipelas" is a very comprehensive one even in Hippocrates; for it is clear that he includes in it various purulent and gangrenous processes occurring on the surface of the body. It appears to have been stretched still farther by the later writers of the school of Cos, who were led by their dogma of the humours to use the term erysipelas as an equivalent of "bilious dyscrasia," and so to speak of an erysipelas of the lungs, erysipelas of the uterus, and so on.⁵ This doctrine, subsequently extended by Galen, was the dominant one throughout all the period following, down to the overthrow of the Galenic system; and in its place there came other theories worked out either from the humoral point of view or from that of solidism. Any account of these developments of the doctrine of erysipelas lies outside the limits of my task, and is of no importance for the historical account of the disease itself.

¹ 'Epidemiorum,' lib. iii, sect. iii, § 3, 4, ed. Littré, iii, 70—76; 'De capitis vulneribus,' § 20, ed. c, iii, 254; 'De vulneribus,' § 9, ed. c, vi, 407.

² Celsus says (lib. v, cap. 26, § 33, ed. Almeloveen, Basil, 1748, 302), "Id autem quod ἐρυσίπελας vocari dixi, solum vulnere supervenire sed sine hoc quoque oriri consuevit." So also Galen, 'Method. med.,' lib. xiv, cap. ii, ed. Kühn, x, 949; Oribasius, 'Synopsis,' lib. vii, cap. 32; Paulus, lib. iv, cap. 21, Basil, 1551, 341. The remark of Aëtius (lib. xiv, cap. 60, ed. Montano, Basil, 1535, iii, 58) is noteworthy: "Saepe enim in cuti tantum diffunditur (scil. inflammatio), carnem ipsam nihil injuria afficiens, idque est quod exquisite erysipelas nuncupatur."

³ Avicenna, 'Canon,' lib. iv, fen. iii, fract. i, cap. 4, 5, ed. Venet., 1564, ii, 109; Ali Abbas, 'Pract.,' lib. iii, cap. xxviii, ed. Lugd., 1523, 194 b.

⁴ Such as the writers of the school of Salerno: Ruggiero, 'Chirurgia,' lib. iii, cap. viii, de erysipelate superveniente vulnere (in de Renzi, 'Collect. Salernit.,' ii, 472); Rolando, 'Morb. med. ratio,' lib. iii, cap. ix; 'Glossulae quatuor magistrorum,' lib. i, p. vi, cap. 23 (in de Renzi, l. c., ii, 559), and Guido (of Chauiac), 'Chir. Tr.,' ii, cap. 3, Lugd., 1572, 64 (following Avicenna, Ali Abbas, &c.).

⁵ No doubt many of the physicians and surgeons of ancient and mediæval times rightly saw that erysipelas occurred mostly in association with wounds. Thus de Vigo says ('Chirurgia,' lib. ii, tract. i, cap. 4, 5, ed. Lugd., 1521, fol. xv): "Accidit etiam ut plurimum in vulneribus a medico male tractatis." The opinion of Tagault ('Inst. chirurg.,' lib. i, cap. 8, in Gessner, 'De Chirurg. script. Tiguri,' 1555, 25), which is adopted almost exactly by Guido, is also noteworthy: "Verum ac legitimum erysipelas raro terminatur suppuratione, sed magna ex parte insensibili transpiratione seu resolutione." From a general survey of the professional writings of that period, it would appear that it was the surgeons who held the most correct views of erysipelas.

In these changing aspects of the term "erysipelas," are reflected the various systems and theories which have dominated the schools of medicine from century to century. The history of the doctrine of the rose (Rothlauf) affords a sort of picture of the development of scientific medicine. And although the most recent inquiries into this disease have departed from the doctrinal point of view and have invested themselves with a character for precision, still we have only to glance at the latest and most important writings on erysipelas,—by Velpeau, Pirogoff, Volkmann, Billroth, Orth, Lukomsky, Tillmanns, Fehleisen and others,—in order to see how far we still are from a common understanding as to what we should include under "Rothlauf," how widely this term may be stretched to embrace inflammatory processes in the skin and subcutaneous tissues, how some would have us think of "erysipelatous affections of the mucous and serous membranes, as well as of other tissues"—in a word, in order to see how the term "erysipelas" has come to characterise a morbid process that is one in its origin but various in its forms.

In seeking for the means of satisfactorily answering the question which we propose to ourselves, we shall have to go to the essential cause of the disease. However widely men's views still diverge as to what we name "erysipelas," or ought so to name, there is no longer any doubt that the disease itself, be the word used in as narrow or as comprehensive a sense as any one pleases, consists in an infective process. If, then, the specifically infective matters be recognised for one of the various nosological forms included under erysipelas, best of all for the so-called "érysipèle légitime," the detection of that specific matter in other forms would justify us in deciding that they belonged to the morbid states comprehended in the notion "erysipelas," just as we might conclude from its absence in still other forms that these latter were outside the meaning of the term.

But although the latest researches on this very "érysipèle légitime" have disclosed facts, as will appear, that are deserving of all attention, yet these are not adequate to the solution of the problem before us; and thus we are left for the present to develop our notion of the disease merely from

the clinical and epidemiological point of view, or out of observations upon and reasonings about the type of the malady in the individual and in the epidemic. From that point of view, which I shall adhere to throughout this chapter, I may define erysipelas to be an infective inflammation-disease of the skin or of one of the mucous membranes near to the external surface of the body (mouth, throat, vagina, &c.), which in all probability proceeds invariably from a solution of continuity or wound, and is characterised by its rapid extension over the surface, and by the infective fever that accompanies the local process; the latter in many cases is confined to the skin, and in such cases mostly heals rapidly, leaving no permanent effects; but in other cases it extends to the subcutaneous (or submucous) tissue, sometimes even to still more deeply situated parts (phlegmonous erysipelas), and leads to more or less considerable suppurations spreading along the surface, or to gangrenous destruction of parts (malignant or gangrenous erysipelas), under certain circumstances even to secondary affections of other (internal) tissues or organs.

§ 148. MOSTLY A DISEASE OF THE TEMPERATE ZONE.

Just as it has been met with at all periods in the history of mankind, erysipelas occurs in all parts of the world—in some, however, more commonly than in others.

The available statistics, which are extremely cumbrous and, for many reasons, not very trustworthy, do not enable us to state in figures the amount of the disease in the various countries or regions of the globe. This much we may conclude from them of a general nature, that the malady has been found to be tolerably uniform in distribution and in the number of cases throughout the *temperate zone of both hemispheres*.

Of the comparatively frequent occurrence of the rose in the polar regions, we have accounts from *Iceland*, the *Faröe Islands*, where it is not unfrequently epidemic,¹ *Greenland*, where it has also broken out repeatedly in great epidemics,

¹ Martius, 'Revue méd.,' 1844, Févr.; accounts in 'Sundhedskoll. Forhandl. for Aaret,' 1846, 13; 1851, 35; 1855, 51.

and where it would appear to be more common in the northern districts than in the southern,¹ and from *Alaska* (New Archangel).²

From warm and subtropical latitudes, we have similar accounts of the prevalence of the disease—from such countries as *Turkey*,³ *Asia Minor*⁴ (the Troad), *Syria*,⁵ *Persia*,⁶ *Egypt*,⁷ *Tunis*,⁸ and *Algiers*.⁹ In *Japan* erysipelas would appear to be very uncommon, for Wernich did not see a single malignant case of it during a residence of several years.¹⁰ Whether the really tropical regions enjoy that remarkable freedom from erysipelas which has often been claimed for them is doubtful; at all events, the information from these countries is to be received with caution. Celle¹¹ says that he did not see ten cases of simple erysipelas during a seven years' residence in *Mexico* (mostly at Mazatlan); Christie¹² knew of only one case in *Zanzibar* in the space of five years; Voigt,¹³ Mackinnon,¹⁴ Huillet¹⁵ and others emphasise the rarity of its occurrence in *India*; Tschudi¹⁶ mentions that it is much commoner in *Peru* in the "puna" region than in the Eastern Sierra. On the other side, Pellissier¹⁷ says that he has seen erysipelas very often in *Réunion*; and it follows from the statements of Annesley¹⁸ and Morehead,¹⁹ that traumatic

¹ Lange, 'Bemaerkn. om Grönlands Sygdomsforhold,' Kjöbenhavn, 1864, 37.

² Blaschke, 'Topogr. med. portus Novi-Archangelensis,' Petropoli, 1842, 65.

³ Rigler, 'Die Türkei und deren Bewohner, &c.,' Wien, 1852, ii, 50.

⁴ Virchow, in his 'Archiv,' 1879, Bd. 77, 174.

⁵ Tobler, 'Beitr. zur med. Topogr. von Jerusalem,' Berl., 1852, 39.

⁶ Polak, 'Wochenbl. der Gesellsch. der Wiener Aerzte,' 1857, Nr. 46, 737.

⁷ Pruner, 'Krankheiten des Orients,' 119.

⁸ Ferrini, 'Saggio sul clima . . di Tunisi, &c.,' Milano, 1860, 185.

⁹ Guyon, 'Gaz. méd. de Paris,' 1839, Nr. 46; Villette, 'Mém. de méd. milit.,' 1842, liii, 125; Burdiat, 'Observ. et réflex. sur des cas nombreux d'érysipèles, &c.,' Montp., 1847 (relating to the camp at Teniet-el-Hâd).

¹⁰ 'Geographisch-med. Studien, &c.,' Berl., 1878, 196.

¹¹ 'Hygiène des pays chauds,' Par., 1848.

¹² 'Brit. Med. Journ.,' 1872, June, 577.

¹³ 'Bibl. for Laeger,' 1833, Heft 3, 2.

¹⁴ 'Indian Annals of Med. Sc.,' 1854, Oct., 177.

¹⁵ 'Arch. de méd. nav.,' 1868, 25. During a residence of several years at Pondicherry, he saw only one severe case of traumatic erysipelas.

¹⁶ 'Oest. med. Wochenschr.,' 1846, 661.

¹⁷ 'Considér. sur l'état des malad. les plus communes à la Réunion,' Par., 1881, 46.

¹⁸ 'Researches into the more prevalent Diseases of India,' Lond., 1841, 544.

¹⁹ 'Clin. Researches on Disease in India,' Lond., 1856, i, 361.

erysipelas is far from rare in *India*, and that it becomes epidemic from time to time under the same circumstances as in other parts of the world.

No weight attaches, however, to the data of Jobim, Rendu,¹ Sigaud² and other authorities for *Brazil* as to the endemic prevalence there of wildfire of the legs; for the disease in those cases is clearly not erysipelas but elephantine dermatitis (pachydermia).

§ 149. ENDEMICS AND EPIDEMICS, MOSTLY IN HOSPITALS.

Erysipelas occurs either *sporadically* or in *epidemics*. The head-quarters of the disease have always been found in self-contained places occupied by a considerable number of persons, most of all in hospitals, where erysipelas is not unfrequently *endemic* for long periods, and, next to them, in lying-in and foundling hospitals, lunatic asylums, educational institutions, ships, and the like. On the other hand, among the people at large, sporadic cases of erysipelas occur comparatively seldom; no doubt from time to time there are cases occurring in groups, forming a kind of epidemic, but it is the exception for these outbreaks to reach any considerable extent, and they sometimes coincide with the epidemics of erysipelas in hospitals.

The literature of medicine is full of references to the *endemics of erysipelas in hospitals*. I may mention, by way of examples, Boinet's³ account for the Hôtel Dieu of Paris, relating to certain notorious wards in particular, that of Wells⁴ for various hospitals in London at the end of last century and beginning of the present, of Fenger⁵ for the Frederiks Hospital at Copenhagen, of Kern⁶ for the University Clinique at Marburg, of Reese⁷ for the Bellevue Hospital of New York, and of a

¹ 'Études topogr. et méd. sur le Brésil,' Par., 1848, 74.

² 'Du climat et des malad. du Brésil,' Par., 1844, 157, 369. Compare the account by da Silva ('Arch. de méd. nav.,' 1880, Mai, 331) of a form of erysipelas and lymphangitis prevalent in Brazil.

³ 'Journ. des conaiss. méd.-chir.,' 1839, Nr. 7.

⁴ 'Transact. of the Soc. for the Improvement of Med. and Chir. Knowledge, 1800, ii, 213.

⁵ 'De erysipelate ambulanti disqu.,' Havn., 1842.

⁶ 'De erysipelate, imprimis epidemico,' Marb., 1845.

⁷ 'Amer. Journ. of Med. Sc.,' 1850, Jan., 98.

writer¹ whose account relates to the hospital at Melbourne. Still more numerous are the accounts of *epidemics* of the disease in *hospitals and lying-in institutions*.² Among the more recent records of hospital erysipelas, I may mention those of Serre for the Hôtel Dieu of Montpellier in 1840, Marjolin and Langier³ for the Hôpital Beaujon in 1843, Billroth⁴ and Waeckerling⁵ for the Zurich Hospital in 1859-60, Bourgeois⁶ for the hospital at Estempes in 1860, Desgranges⁷ and Ollier⁸ for the Hôtel Dieu of Lyons the same year, Fenestre⁹ for the Hôpital Beaujon of Paris also the same year, Pujos¹⁰ for the Hôpital St. André at Bordeaux in 1863, Ponfick¹¹ for the Heidelberg Surgical Clinique in 1866, Ollier¹² for the Hôtel Dieu of Lyons in 1867, Volkmann¹³ for the Halle Surgical Clinique in 1868, Savory¹⁴ for St. Bartholomew's Hospital (London) in 1872-73, and Miller¹⁵ for the Edinburgh Infirmary in 1879-80.

As regards *epidemics of erysipelas in lunatic asylums*, we have an account by Rayer of one in Paris in 1828; in *educational institutions*, the paper of v. Nymann¹⁶ relating to Smolna; *on board ship*, an account by Busk¹⁷ of an endemic on the hospital ship "Dreadnought" in 1837-38, as well as notices of its prevalence in the British Mediterranean fleet¹⁸ in 1852, and one by Smart¹⁹ on the epidemic of 1824 in Devonport dockyard and on that of 1873-74 in Portsmouth dockyard. Instances of extensive epidemics of erysipelas occurring elsewhere than in such places are given by the following writers:—Black²⁰ for the epidemic at Bolton in 1832, Wutzer²¹ for the disease at Bonn in 1849, Alison²² for the erysipelas of 1850 in Edinburgh, Deutsch²³ for that of 1856 in the

¹ 'Med. Times and Gaz.,' 1871, March, 287.

² For the epidemics of erysipelas in lying-in hospitals, see the chapter on "Puerperal Fever."

³ 'Arch. gén. de méd.,' 1846, Debr., 414.

⁴ 'Arch. für klin. Chir.,' ii, 460.

⁵ 'Deutsche Klin.,' 1861, Nr. 19.

⁶ 'Journ. des connoiss. méd.-chir.,' 1861.

⁷ 'Gaz. méd. de Lyon,' 1861, Juin.

⁸ Ib., Août.

⁹ 'Sur une épidémie d'érysipèle, &c.,' Par., 1861.

¹⁰ 'De l'érysipèle épidémique,' Par., 1865.

¹¹ 'Deutsche Klin.,' 1867, Nr. 20 ff.

¹² 'Lyon médical,' 1868, Nr. 37.

¹³ 'Handb. der Chir.,' by Pitha-Billroth, i, 2 Abth., 153.

¹⁴ 'Brit. Med. Journ.,' 1873, Jan., 5.

¹⁵ 'Edinb. Med. Journ.,' 1880, June, 1095.

¹⁶ 'Arch. für Kinderheilkde.,' 1880, i, 466.

¹⁷ In Nunneley, 'Treatise on the Nature . . of Erysipelas,' Lond., 1841, 146.

¹⁸ 'Statist. Reports,' 1853, 122.

¹⁹ 'Brit. Med. Journ.,' 1880, Feb., 200.

²⁰ 'Transact. of the Prov. Med. Assoc.,' 1837, v, 203.

²¹ 'Rhein. Monatschr. für pract. Aerzte,' 1849, Sptbr., Octbr.

²² 'Edinb. Monthl. Journ. of Med.,' 1851, Jan., 72.

²³ 'Preuss. med. Vereins-Ztg.,' 1857, Nr. 49.

Circle of Pless, Upper Silesia, the reporters¹ on an epidemic in several communes of the Lower Rhine in 1858, Lange² for the disease in North Greenland, 1861, Pujos³ for the epidemic of 1863 in Bordeaux and several other places in the Gironde, Dechambre⁴ for the disease in Paris at the same time, the reporters⁵ for several counties in Pennsylvania in 1866 and 1872-73, Tibbits⁶ for the epidemic of 1873 at Bristol, Radcliffe⁷ for Oxford in 1874, and Baader⁸ for the village of Buus (Canton Neuchâtel) in 1875-76.

§ 150. EPIDEMICS COMPLICATED WITH THROAT-AFFECTION.

Besides the affection of the skin, there occur in the course of the erysipelatous process certain lesions of organs; and of these the one that has most interest for our inquiry is the *inflammatory affection of the mucous membrane of the throat*, which appears either primarily or secondarily during the attack. It consists either in simple catarrh or in phlegmon, or it assumes the so-called diphtheritic character, that is to say, a tendency to necrosis; and it may spread from the throat to the larynx, giving rise to symptoms of œdema of the glottis or of laryngeal croup. In correspondence with the severity of the local lesions under those circumstances, there is an aggravation of the constitutional symptoms due to general infection; secondary foci of disease may even arise in the internal organs (meninges, lungs, intestine, &c.), and the malady may acquire a "typhoid" character which will warrant us in speaking of it as *erysipelas typhoides* or *erysipelas malignum*.

This malignant form of erysipelas occurs either sporadically (not rarely during epidemics of ordinary erysipelas of the skin) or it may occur in groups of cases or even in considerable epidemics. One of the earliest references of the latter kind will be found in Darluc's⁹ account of an epidemic of erysipelas

¹ 'Trav. du conseil département d'hyg. publ. du Bas-Rhin,' 1865, ii, 2.

² L. c.

³ L. c.

⁴ 'Gaz. hebd. de méd.,' 1863, Nr. 30.

⁵ 'Transact. of the Pennsylvania State Med. Soc.,' 1867; 1873, 129, 169, 174; 1874, 226.

⁶ 'Lancet,' 1864, June, 832.

⁷ 'Brit. Med. Journ.,' 1875, May, 651.

⁸ 'Correspondenzbl. für Schweiz. Aerzte,' 1877, Nr. 3 ff.

⁹ 'Journ. de méd.,' 1757, Juill, vol. vii, 55.

observed by him at Caillan in the summer of 1750, in which the rose in the face was followed in many cases by a severe affection of the throat or larynx, the patients dying with symptoms of an "esquinancie funeste." The same type presented itself in the epidemic at Montrose in 1822, of which Gibson¹ says: "The disease was not so much confined to the head or face, as common erysipelas, but it frequently attacked other parts of the surface of the body. Sometimes the internal fauces were attacked, and if it spread to the trachea it generally proved fatal."

Other instances are the epidemic of 1833-34 in Dublin, described by McDowell,² and that of 1847 in London, reported on by Gull and Lever;³ also the hospital erysipelas which was prevalent in 1870-71 in the military ambulance at Berlin, where cases of erysipelas of the face complicated with catarrhal, phlegmonous or diphtheritic angina, were observed by Hesse and Hiller.⁴ Sporadic cases of the same kind have been reported by Stevenson,⁵ Arnott,⁶ Simon,⁷ zum Sande,⁸ Heubner,⁹ Jacobs¹⁰ and others. In a report of the surgical clinique at Greifswald for the year 1876, Schüller says:¹¹

"The coincidence with erysipelas of diphtheritis of wounds and mucous membranes, which had formerly been noticed from time to time, appears to have been regarded as something more than an accident, even although no particular importance was attached to it. Now-a-days that phenomenon is justly regarded with greater attention. In such cases we are very often able to make out a diphtheritic process, either proceeding directly from the erysipelas or arising during the existence of the latter, and attacking not only the wound but also the mucous membrane of the mouth and throat. Hardly less interesting [than the coincidence of the diphtheritis of wounds with erysipelas] is the observed correspondence between erysipelas and diphtheria of the

¹ 'Transact. of the Edinb. Med.-Chir. Soc.,' 1828, iii, 94.

² 'Dublin Quart. Journ. of Med. Sc.,' 1834, Nov., 161.

³ 'Med. Gaz.,' 1849, June.

⁴ 'Deutsche med. Wochenschr.,' 1876, 309, 323.

⁵ 'Transact. of the Edinb. Med.-Chir. Soc.,' 1826, ii, 128.

⁶ 'Lond. Med. and Phys. Journ.,' 1827, March, 194.

⁷ 'Arch. gén. de méd.,' 1865, Oct.

⁸ 'Journ. für Kinderkr.,' 1871, lvii, 57.

⁹ 'Jahrb. für Kinderheilkde.,' 1872, vi, 105.

¹⁰ 'Presse méd. belge,' 1875, Nr. 16.

¹¹ 'Deutsche Zeitschr. für Chirurgie,' 1877, viii, 540 ff.

mucous membrane of the mouth and throat. The latter, no doubt, has its prototype in the pharyngeal catarrhs which very often accompany erysipelas. But the same complication has been observed in more considerable epidemics, one of which in North America is described by Hirsch."

The concluding reference here is to those epidemics of erysipelas in the Western Hemisphere, particularly in the United States, with complications of severe throat affection and distinguished by so-called typhoid phenomena, to which I directed the attention of the profession in Germany in the first edition of this work, and which constitute one of the most interesting episodes in the history of erysipelas.¹

§ 151. REMARKABLE SERIES OF EPIDEMICS IN NORTH AMERICA.

The first accounts of these epidemics of malignant or typhoid erysipelas in the Western Hemisphere date from the years 1822–1836 (see the tabular survey at p. 401). From 1841 onwards the disease grew into a pandemic which did not cease until the beginning of the sixties. The subsequent accounts from that part of the world relate to more isolated epidemic outbreaks.

The type of the disease may be gathered from the following account, which has been compiled from the best sources.²

Clinical characters.—The disease broke out either suddenly with febrile manifestations, or it developed gradually. In either case the patient complained, before there was any affection of the skin or throat, of extreme weakness, of pains in the head, back and limbs, and of sickness. Shortly after, pain in swallowing began to be noticed,

¹ Volkmann is inclined to think that in these epidemics we have to do with "an affection very closely related to diphtheritis, perhaps even with pure diphtheritis of the throat." From the point of view of morbid anatomy, this opinion is perfectly justified; but from the clinical and etiological points of view I am unable to agree with it, the less so that the profession in North America, at the time when the observations on these epidemics were made and recorded, were very well acquainted with angina maligna or diphtheritic angina. *I reserve the more particular defence of my views on the significance of "diphtheria" until the chapter on Angina maligna (or the so-called diphtheria of the throat).*

² The references to authorities are given in one of the columns of the chronological table.

being the earliest characteristic symptom; an examination showed swelling of the tonsils and mucous membrane of the throat, which, in the milder cases, were red and œdematous, and afterwards looked to be covered by a layer of purulent mucus; while, in the more severe developments of the malady, the mucous membrane of the pharynx was seen to be reddened or of a dark purple colour, which extended gradually to the gums, the tongue, and the side of the cheeks, the tongue itself swelling greatly and assuming a dark brown colour—whence the popular name of “black tongue.” In such cases the mucous membrane, especially where it is reflected over the gums and alveolar process, was often found covered with ash-coloured sloughs; when these were thrown off, deep ulcers remained behind, which made the act of swallowing extremely painful. Sometimes the pharyngeal inflammation extended to the larynx and trachea, when the symptoms of croup would supervene; at other times it extended to the nasal passages, and thence to the frontal sinuses and even to the antrum. These manifestations, which were almost always accompanied by swelling of the lymphatic glands of the neck, sometimes by lymphangitis or severe neuralgias of the temporal and occipital regions, lasted usually until such time as the exanthem appeared on the skin, which was generally within the first two days, but sometimes not until later. In many cases there was only a slight development of erysipelas, and in some it appears to have been absent altogether, the patients suffering from the throat affection only. The outbreak of erysipelas was usually indicated by tension, heat, and stinging pains in the affected part; in a short time the skin looked to be more or less swollen, and of a dark or bright red according as the lesion was deeply seated or on the surface. If the upper layers of the skin only were involved, the course of the disease was usually that of simple erysipelas; but if the subcutaneous tissue were also implicated, the result was often deep and widespread ichorous and gangrenous destruction of tissue. In many cases it was the connective tissue that was first attacked, particularly in the axilla, where the destructive process would not unfrequently extend to the muscles and lymphatic glands; so that shreds of exfoliated connective tissue, fragments of lymph-glands, and the like were discharged with the ichor when the abscess was opened, and muscles or even bones were laid bare. The ichor that was let out was so strongly corrosive that it attacked the hardest steel as if it had been nitric acid, and instruments which had been used to open the abscess and left lying for some hours without being cleaned, were completely eaten into and rendered useless. Another and more frequent issue of the deep process was gangrene, which sometimes set in on the shortest notice, and would destroy the whole substance of a limb, and in some cases the soft parts of the whole of one side of the face. Although the erysipelas occurred in all parts of the body, its chief seat was the face, where it usually began about the wing of the nose or the canthus of the eye, extending upwards to the scalp and downwards to the neck and shoulders, and sometimes even over the whole body. The deeper collections of matter, also, were found

in all parts of the body; in one case Bennet saw them begin to form in the axilla and gradually spread over most of the trunk.

Besides those two strictly pathognomonic conditions, many other local affections occurred in the course of the malady, but not so uniformly: such as bronchitis and pneumonia, pleurisy, meningitis, peritonitis (an almost constant symptom of the disease in Greenland, and of the epidemic in Montgomery County in the spring of 1852), and affections of the intestinal mucous membrane (revealed in life by vomiting and purging), or of the genito-urinary system, in which case suppression of urine and bleeding from the urethra were not unfrequently observed.

The fever, in well-developed cases, had always a typhoid character. It must appear remarkable that the rigors observed at the beginning of the disease should have recurred more than once during its course without any obvious connexion with exacerbations of the fever. Various degrees of the malady could be distinguished according to the more or less intense development of the complications above sketched. Not unfrequently it was of so mild a type that the patient hardly required medical treatment; another time it would be so malignant that death occurred on the third or fourth day. In cases of a favorable type, the attacks of angina ceased with the appearance of the erysipelas; a few days later the exanthem faded, desquamation set in, and with it there was not unfrequently a formation of numerous small abscesses under the skin, which healed quickly, however, under simple treatment. When the type of the attack was unfavorable, the exanthem assumed a bluish colour, the skin and connective tissue sloughed, large burrowing abscesses formed having the surviving tracts of connective tissue for their basis, the pulse was small and quick, the tongue looked as if coated with a dark brown dry crust, the urine was scanty, diarrhœa and delirium supervened, and death took place usually within ten days of the first appearance of the exanthem. Just as rapid, and almost as fatal, was the course of those cases in which signs of inflammation appeared in the respiratory organs, the meninges, &c., death taking place nearly always within the first eight days, and in most cases within the first six. Finally, in the class of cases where considerable centres of suppuration had developed in the connective tissue, the disease was often protracted many months; and, even when convalescence went on well, there would often be most important functional irregularities, atrophy of muscles, and the like remaining over.

Morbid anatomy.—As to the state of parts found in those who died of this disease we have only a few meagre notices.¹ The most complete are those by Nathusius, who had opportunities of examining the bodies of three negroes after death from typhoid erysipelas. The bodies always

¹ On the very first appearance of the disease several physicians, who had examined the bodies of persons dead of typhoid erysipelas, were seized with it and died; and it was their solicitude to escape the same fate that restrained the United States' practitioners, who were not generally indifferent to the morbid anatomy, from making *post-mortem* examinations in this disease.

looked emaciated, with no blood in them, the skin and connective tissue at the places where the exanthem had been observed during life infiltrated with a gelatinous exudation, which extended into the connective tissue of the nearest muscles and glands, the membranes of the brain and spinal cord full of blood, some bloody serum in the ventricles of the brain and in the pericardium, the heart relaxed and containing a soft black clot in the ventricle, the bronchi injected and full of mucus, the lungs full of blood (hypostatic pneumonia), serous fluid in the pleura and peritoneum, the mucous membrane of the stomach and intestine ecchymosed, the vicinity of the Peyer's patches in the lower end of the ileum congested, but never any ulceration, the mesenteric glands swollen, the liver full of blood, and the spleen soft and friable. The state of the kidneys is not mentioned. Dexter and Hall in one case found the liver softened, the peritoneum injected and containing a dark coloured ichorous fluid with shreds in it, and the coils of inflamed intestine slightly glued together. Bennet¹ observed, in another case, the costal pleura on the right side injected and sticking to the visceral layer by means of recent adhesions, the pulmonary pleura of the left side everywhere deep red with eight ounces of sero-sanguineous fluid in the cavity, the substance of both lungs normal. In a second case, which presented no signs of intestinal affection during life, he found the follicles in the lower end of the ileum very prominent, especially near the ileo-cæcal valve, but none of them were ulcerated; mesenteric glands and spleen perfectly normal.

Chronological Table of all the known Epidemics of Malignant Erysipelas in America from 1822 to 1881.

Epidemic.			Authority.
Time.	Place.		
1822	Spring	Nova Scotia, New Brunswick	Bayard, New York Med. Journ., 1831, May, 54.
1826	...	Jamaica (Kingston).....	Leon, New York Med. and Phys. Journ., 1827, April.
"	Winter	Vermont (Burlington).....	Drake, Treat. on the Principal Diseases of the Interior Valley of N. America, Philad., 1854, ii, 623.
1832	...	New York (Ogdensburg)...	Drake, l. c.
1833	Spring	Ohio (St. Clairsville)	Drake, l. c.
1836	Winter	Ohio (Preble County).....	Drake, l. c.
1841	...	Ohio	Holston, Transact. of the Ohio State Med. Soc., 1857.
"	Summer	East Canada	Drake, l. c.

¹ 'New York Journ. of Med.,' 1853, July 20, p. 23.

Epidemic.		Authority.
Time.	Place.	
1841-2	Winter	Vermont (Middleburg) ... Drake, l. c.
"	"	New York (Moriah)..... Drake, l. c.
1842	Spring	Vermont (St. Albans)..... Hall and Dexter, Amer. Journ. of Med. Sc., 1844, Jan.
"	Autumn	Diffusion along Lake Champlain and the Connecticut River
"	November	New York (Cortlandville and other centres) Shipman, New York Journ. of Med., 1846, Jan., 25.
1843	Winter	Indiana (Ripley County), in considerable diffusion Sutton, Western Lancet, 1843, Nov., 308.
"	Spring	Missouri (various places) . Bennet, Western Journ. of Med., viii, 110.
"	"	Ohio (Miami Valley) Drake, l. c.
"	"	North Greenland..... Accnt. in Sundhedscolleg. Forhandl. Aaret, 1844, 57.
"	November	New York (Erie County).. Jewett, Buffalo Med. Journ., iii, 262.
"	December	Indiana and Michigan..... Shipman, l. c.; Meeker, Illinois Med. and Surg. Journ., 1844, June.
1843-4	Winter	Canada (Montreal) } Drake, l. c.
"	"	New York (various places) } Drake, l. c.
"	"	Illinois (Blumington)..... } Drake, l. c.
"	"	Ohio (Montgomery County) Carey, Transact. of the Amer. Med. Assoc., 1854, vi, 310.
"	"	Wisconsin (Milwaukie) ... } Drake, l. c.
"	"	Kentucky (Louisville)..... } Drake, l. c.
"	"	Only in the Hospital
"	"	Mississippi (various places) }
1844	Spring	North Greenland..... Sundhedscolleg. Forhdl. for Aaret, 1845, 37; Kayser, Ugeskrift for Laeger, 1846, Nr. 15, 229.
"	"	Pennsylvania (Delaware County) Young, Amer. Med. Examiner, 1844, Sept.
"	"	Tennessee (Memphis, Columbia) Shanks, Western Journ. of Med., iii, 12; Robard, ib., iv, 285.
"	"	Mississippi (Grand Gulf) . Drake, l. c., 628.
"	Winter	New York (Livingston Co.) Drake, l. c.
"	"	Indiana (Laporte) Shipman, l. c.
"	"	Missouri (St. Louis and vicinity) Moore, Missouri Med. and Surg. Journ., ii, 97.
"	"	Virginia (Petersburg)..... Peebles, Amer. Med. Journ., 1846, Jan., 23.
1845	February	Alabama (Courtland) Drake, l. c.
"	Spring	North Greenland..... Sundhedscolleg. Forhdl. for Aaret, 1846, 15.

Epidemic.			Authority.
Time.	Place.		
1845	Spring	Indiana (Logansport).....	Fitch, Illinois and Indiana Med. and Surg. Journ., i, 1.
„	Autumn	New York (Ontario and Erie County)	Drake, l. c.; Pelt, Buffalo Med. Journ., i, 193.
„	„	North Carolina (Elizabeth, Raleigh)	Nathusius, De erysipellate typh. Diss. Berol., 1856; McKee, South. Med. Report, ii, 410.
1845-6	Winter	Pennsylvania (Uniontown)	} Drake, l. c.
„	„	Ohio (Meigs County)	
1847	Spring	Michigan (various places)	Drake, l. c.; Pitcher, Transact. of the Amer. Med. Assoc., 1853, v.
„	Autumn	Connecticut (Bridgeport)	Bennett, New York Journ. of Med., 1848, May, and Amer. Journ. of Med. Sc., 1850, April, 377.
1847-8	Winter	Pennsylvania (various places)	Corson and Geiger, Transact. of the Pennsylvania State Med. Soc., 1848.
1848	...	New Jersey	} Accounts in Transact. of the Amer. Med. Assoc., 1850, ii.
„	...	Connecticut	
„	...	Ohio	
„	February	Mississippi (Jackson)	Farrar, Southern Med. Reports, i, 355.
1848-9	Winter	Connecticut (Hartford County)	Russel, Proc. of the Connecticut State Med. Soc., 1855.
„	„	Ohio (Brown County).....	} Drake, l. c.
„	„	Mississippi (Vicksburg)...	
„	„	Louisiana (New Orleans). Only in the Hospital	
1850	Spring	Massachusetts (Boston, a few cases)	Morland, Amer. Journ. of Med. Sc., 1850, Oct., 318.
„	„	Pennsylvania (Armstrong County)	Gillespie, Amer. Med. Examiner, 1851, March.
1851	„	Michigan (Detroit)	Pitcher, l. c. (ad ann. 1847).
„	„	Pennsylvania (Blair Co.)	Rodrigue, in Transact. of the Pennsylvania State Med. Soc., 1852.
„	Summer	California (Sacramento)...	Blake, Amer. Journ. of Med. Sc., 1852, July, 59.
1851-2	Winter	Pennsylvania (various places)	Accounts in Transact., l. c. (ad ann. 1851); Leasure, Amer. Journ. of Med. Sc., 1856, Jan., 45.
„	„	Ohio (Shelby County).....	Carey, l. c.
1852	...	Ohio (Montgomery County)	Ib.
„	...	Kentucky (Bordstown) ...	Mattingly, St. Louis Med. and Surg. Journ., 1853, May, 217.

Epidemic.			Authority.
Time.	Place.		
1852	January	Missouri (Platte County, sporadic cases)	Ridley, New York Journ. of Med., 1853, Jan., 41.
1853-4	Winter	Ohio (Highland County)...	Carey, l. c.
1854	...	Pennsylvania (Montgomery County)	Corson, l. c.
,,	February	New York (Venango Co.)	Avery, Transact. of the New York State Med. Soc., 1855.
1864	...	Pennsylvania (very widely spread)	Transact. of the Pennsylv. State Med. Soc., 1864.
,,	Winter and Spring	Illinois (Birmingham, Chicago, Waverly, and various other places)	King, Amer. Journ. of Med. Sc., 1865, Jan., 274; accounts by Davis in Transact. of the Illinois State Med. Soc., 1864; McVey, ib., 1865.
1866	...	Pennsylvania (Lehigh Co. and others)	Accounts in Transact. of the Pennsylv. State Med. Soc., 1867.
1870	Autumn	Minnesota (Ramsey Co. and other places)	Hand, in Transact. of the Minnesota State Med. Soc., 1871.
1880	...	Wisconsin and other States in the North-west	Meachem, Transact. of the State Med. Soc. of Wisconsin, 1881.

This tabular survey presents a picture of the distribution and the epidemic outbreaks of the disease in North America, which is unquestionably a very incomplete one; and its incompleteness is borne out by the language of Holmes¹ in 1854: "Viewed as an epidemic, the disease demands attention from physicians in the West. We suppose small-pox or cholera would not cause greater consternation in many neighbourhoods, than the appearance of that violent type of erysipelas known, from a prominent sign, as the "black tongue;" but, even when unaccompanied by this feature, the disease is much dreaded in the Western States. We believe that it is a disease more common in the West than in the East; and from what we have seen and read and heard of it, we are disposed to think that no part of the earth has suffered more from epidemic erysipelas than Illinois, Indiana, Missouri, and parts of Tennessee and Iowa. There is scarcely a year, or season, in which you may not hear of several centres of the disease in these States; Michigan, Wisconsin and Minnesota Territory, have also suffered much from it. It has raged with great violence on the plains on the route to California; has been very common and of grave type in

¹ 'Transact. of the Amer. Med. Assoc.,' 1854, xv, 155.

Santa Fé; and in California it is a frequent and much dreaded disease. In wet and cold spring months, it is common in Louisiana and Texas; it extends as an epidemic from Maine to Mexico, from Minnesota Territory to Florida."

This statement accords with all the information that I have been able to gather, so far at least as relates to the period during which the disease had been most severe in North America. At the same time it bears out the result that we arrive at from the foregoing detailed table of the epidemics in regard to the *predominance of malignant erysipelas in the Western States*. Of the 70 epidemics above mentioned, 3 were prevalent in Greenland (all 3 in North Greenland),¹ 1 in Nova Scotia and New Brunswick, 2 in Canada, 7 in the New England States (Vermont, Massachusetts and Connecticut), 18 in the eastern central States (New York, New Jersey and Pennsylvania), 5 in the middle central States (Virginia, Kentucky, Tennessee, North Carolina), 26 in the Western States (Ohio, Indiana, Illinois, Michigan, Wisconsin, Missouri, Minnesota), 6 in the Southern States (Alabama, Mississippi, Louisiana), and 1 in California. The epidemic observed at Kingston, Jamaica, in 1826 stands by itself. The extent to which the disease spread in the several localities has been very various; here and there the cases occurred in more or less considerable groups, sometimes in hospitals only; at other places the diffusion was general. Equally various was the duration of the several epidemics, which in some instances amounted to a few months, and in others exceeded a year. Regarded as a whole, the incidence of malignant erysipelas gives us the impression of a series of small and circumscribed epidemics rather than of pandemic prevalence, such as is proper to cholera, the acute exanthemata, and other spreading diseases.

§ 152. INFLUENCE OF CLIMATE.

The fact that erysipelas is distributed over the whole globe proves that *influence of climate* has at least no essential significance for the occurrence of the disease. At the same

¹ See § 148.

time we may conclude with a good deal of certainty that it is more rarely met with in lower latitudes, at least that form of it which is independent of hospitals. The same relation comes out in the epidemic incidence of malignant erysipelas in the Western Hemisphere, where the disease has been much more frequent and much more extensive in the north-western and northern States of the American Union than in the central and southern.

§ 153. INFLUENCE OF SEASON AND WEATHER.

Further support of this idea will be found in the fact that the amount of the sickness shows a dependence on the *season of the year* corresponding in a measure to the relation to climate. The larger number of observers are of opinion that erysipelas occurs more frequently in the colder than in the warmer months of the year; this is the conclusion of Haller from ten years' observations in the General Hospital of Vienna, of Eschbaum¹ from the experience of fifteen years (1865—1879) in the clinique and policlinique of Bonn, of Boinet and other Parisian hospital physicians and surgeons for the Hôtel Dieu, of Gosselin from seven years' observation in the Hôpital Beaujon, the Charité and the Pitié in Paris, of Pujos from the practice of the Hôpital St. André in Bordeaux, of Borbone² from the statistics of the Turin hospital, of Copland³ and Doig⁴ on the basis of observations made in London, of v. Nymann from the experience of the Educational Institute at Smolna, of Ucke for Samara, Polak for Persia, Annesley for India, and of Reese⁵ and an anonymous authority⁶ from eleven years' (1861—1871) observations in the Bellvue Hospital of New York. Also in the North American epidemics of malignant erysipelas, the greatest incidence of the disease has been very distinctly in

¹ 'Beitr. zur Statistik einiger acut entzündl. und Infections-krankheiten,' Bonn, 1880, p. 20.

² 'Giorn. della Acad. di med. di Torino,' 1878.

³ 'Dictionary of Pract. Med.,' iii.

⁴ 'Med. Times and Gaz.,' 1862, Sept., 72.

⁵ 'Amer. Journ. of Med. Sc.,' 1850, Jan., 98.

⁶ 'New York Med. Record,' 1872, Sept., 373.

the cold months, as may be seen in the foregoing table of them.

The connexion, then, of erysipelas with the colder seasons of the year, which, if not an absolute one, is at least remarkably uniform, may be explained by the direct influence of the *kind of weather*, the weather, namely, which is proper to autumn and early spring. At all events many observers are agreed that considerable fluctuations in temperature, and more particularly the change to cold and damp weather, help materially to produce the disease; and in like manner several of the authorities are of opinion that the epidemics in North America were most prevalent in times of cold and damp. We must regard it for the present an open question how this pathogenetic influence of the weather is to be interpreted—whether it increases the predisposition of individuals to become ill, or whether it stands in some relation to the morbid cause, favouring the origin and diffusion of the disease itself. Doubtless we have here to consider also the influence which the season exerts on the people's mode of life, a subject which I shall have occasion to inquire more fully into in the sequel. Meanwhile, in framing an opinion on the question before us, it should be kept in mind that erysipelas has occurred not unfrequently in summer, and that it has spread to a considerable extent during dry and warm weather, and even during heat, as at Devonport¹ in 1824, Paris² in 1863, in Algiers³ in 1844, and in New Brunswick in 1822.

§ 154. ALLEGED INFLUENCE OF DAMP SOIL.

I hold it to be very doubtful whether *states of the ground*, such as elevation, geological characters, moisture and the like, have any real significance for the occurrence of erysipelas. As in almost all the infective diseases of late, so in erysipelas special importance for the pathogenesis has been assigned to the copious saturation of the ground, or, in other words, to its swampy character. Thus Boinet points

¹ Smart, l. c.

² Duchambre, l. c.

³ Burdiat, l. c.

out that the Hôtel Dieu is built close to the Seine, whose sluggish stream at that point produces excessive saturation of the ground and contributes to the development of putrid effluvia: "J'ai remarqué encore," he says, after giving a detailed statement of the facts, "qu'il y avait une certaine coïncidence entre les grands crues de la Seine et l'apparition des érysipèles. Toutes les fois que la Seine s'accroît, elle se répand dans les caves de l'Hôtel-Dieu, et l'eau y séjourne pendant six semaines, deux mois, plus ou moins; alors et pendant tout le temps qu'elle met à s'écouler, on voit régner ces érysipèles et tous les malades qui ont subi de grandes opérations succombent." But when the water falls and the ground is left dry, on the season of warmth and drought setting in, an abatement of the disease sets in also. Annesley remarks of the incidence of erysipelas in the hospitals of India that "this occurrence is most frequently observed to take place in the more marshy and unwholesome situations near the mouths and banks of rivers;" and other hospital physicians and surgeons have published similar observations to prove the great frequency of erysipelas in buildings erected on damp sites. Further, some of the North American practitioners have believed themselves warranted in assuming that a marshy soil favoured the production and diffusion of the malignant erysipelas on that continent; thus Nathusius pointed out that the disease was prevalent in the swampy districts of North Carolina and adjoining parts of Virginia, and the chroniclers of the epidemic at Burlington, Vt., call attention to the situation of the town on the shore of Lake Champlain.

The influence of this factor upon the origin and propagation of the disease in hospitals cannot be made out with any kind of trustworthiness, inasmuch as a good many other etiological factors have to be taken into account along with it; and it remains accordingly a question what importance, if any, the wetness of the soil assumes among them. It is at least certain that small and clean-kept hospitals have seldom or never been visited by erysipelas, a damp situation notwithstanding; whereas there are many large hospitals, on a dry site but under the converse conditions of hygiene, that have never been altogether free from the disease, and have

often had it in the form of an epidemic lasting for months, although there had been no corresponding change in the state of the ground.

This alleged influence of a swampy soil on the outbreak and spread of malignant erysipelas in North America has been absolutely rejected for good reasons by the great majority of observers. "This supposition," say Hall and Dexter,¹ "is disproved by the fact of remote situations; the hilly regions of the interior, secluded from any emanations of moisture, being alike obnoxious to the influence of this fatal scourge." In Pennsylvania it has been just the hilly districts that have been affected oftenest and most severely. From the facts that he had collected relating to the distribution of the disease, Drake² concludes: "Its victims dwelt upon rocks of every kind, on granite, limestone, sandstone, slate and clay, and of every geological age, from the primitive to the alluvial; it occurred on mountain slopes, low hills, and flat bottom-lands;" and the same conclusion has been come to by Bennet, Sutton and others.

§ 155. NO RACIAL IMMUNITIES.

According to the experience of Rigler in Turkey, Polak in Persia, Pruner in Egypt, and Tschudi among the Indian population of Peru, the various *races and nationalities* would appear to be about equally predisposed towards erysipelas. Thevenot's assertion that the negro race enjoys immunity from erysipelas has been absolutely contradicted by Chassaniol;³ Farrar and Nathusius, also, have seen malignant erysipelas in the negroes, although less commonly, as the latter remarks, than in mulattoes and whites, the proportions being as 10 to 15 and 20.

With reference to the type of the skin-affection in the coloured races, Rigler says that the skin assumes a copper colour in the brown races, whereas in negroes the presence of an erysipelas is shown merely by rise of temperature, by swelling which pits on pressure, and by subsequent desquamation of the cuticle. The new-formed epidermis appears

¹ L. c., p. 21.

² L. c., p. 625.

³ 'Arch. de méd. nav.,' 1855, Mai, p. 514.

for a time less dusky than at other parts of the skin, but it shortly acquires the original look in consequence of abundant deposition of pigment.

§ 156. BREAKS OUT BOTH IN INSANITARY AND CLEAN HOSPITALS.

An importance not to be underrated in the production and diffusion of erysipelas attaches to *unhygienic conditions*, to the accumulation of products of decomposition and putrefaction in dwelling-houses, to the overcrowding of the same with no regard to their cleanliness and ventilation, and to the entrance of sewer gases or of solid particles in the form of dust from cesspits and the like. It is obvious that these nuisances have a quite special influence in hospital wards, particularly in those set apart for the reception of surgical cases; and this is the reason or part of the reason why the disease is particularly often endemic or epidemic, as the so-called “erysipelas nosocomiale,” on the surgical side of large and much frequented hospitals. The greater prevalence of erysipelas in the cold season, which we have already referred to, may not improbably be connected with the same factor in the causation, inasmuch as it is often difficult at that time of the year to secure a continuous and sufficient ventilation of the wards. The literature of medicine is rich in observations which establish the importance of this etiological factor beyond doubt, proving in fact, that the disease has been obviated either permanently or for considerable periods when those nuisances which had called forth the endemics or epidemics of it had been removed.

Summing up the experience of hospital physicians and surgeons in England, Gregory¹ enumerates the following as material causes of hospital erysipelas: overcrowding of the wards, particularly with patients suffering from extensive suppurating wounds or discharging other morbid secretions to a large amount; insufficient cleansing of beds, mattresses, walls, &c.; inadequate ventilation of the wards. This is the conclusion to be drawn also from the observations of Doig, Erichsen,² Campbell de Morgan,³ and other English surgeons, and from

¹ ‘Lectures on the Eruptive Fevers,’ Lond., 1843.

² ‘Brit. Med. Journ.,’ 1874, Jan., 134.

³ In Holmes’ ‘System of Surgery,’ 2nd ed., Lond., 1869, i, 206.

those of Fenger¹ at the Frederiks-Hospital of Copenhagen. Erichsen, who gives his own experience at University College Hospital in the autumn of 1872 in support of his opinion, says: "that erysipelas is often of epidemic origin, there can be no question, but the influence of any epidemic is immensely increased by an unhealthy condition of a ward from overcrowding." Campbell de Morgan observes that, at the Middlesex Hospital, erysipelas, which used to be very common, became much rarer when the sanitary condition of the hospital was improved, particularly through the adequate ventilation of the wards; if patients were admitted with erysipelas, it no longer spread from them to other patients, as it used to do. For the same hospital, Thomson² gives the following interesting fact: In one of the largest wards on the ground floor, cases of erysipelas occurred time after time for a number of years in two beds placed one on each side of a window. On inquiry it was found that the common dust-hole of the hospital was situated in a vault in the western front area exactly under this window and that it had been left open, the effluvia from it entering through the window and particularly affecting the two beds in question. Great care was taken to have the dust-hole thoroughly covered in, whereupon the endemic came to an end for a time. After a considerable period, cases of erysipelas again began to occur in those two beds, when it was found on inquiry that the dust-hole had been again left open. The nuisance was taken away altogether, and no more cases of erysipelas were seen in that ward for a number of years. In the Radcliffe Infirmary at Oxford a severe epidemic of erysipelas occurred in the summer and autumn of 1874, which owed its origin, as Netten Radcliffe³ showed, to conditions of the same kind but worse in degree, namely, the entrance of sewer gas from drains blocked with faecal matters. In the surgical division of the hospital at Rostock, König⁴ had occasion to follow a small epidemic of erysipelas, whose origin could be traced to the pillows used on the operating-table being covered with dried blood-stains; during a certain period every operation done on that table had been followed by erysipelas; but on the changing the pillows the endemic ceased all at once.

However highly we may rate the importance of the noxious influence in question, for producing erysipelas and causing it to spread, it is in something else that we have to seek for the true and essential morbid factor. "Even in hospitals that are admirably constructed," says Volkmann, "that have been made salubrious to the point of luxury, and are perfectly ventilated—even in these there have occurred epidemics of erysipelas of the severest kind; and the most

¹ 'De erysipellate ambulanti disqu,' Havn., 1842.

² 'Med. Times and Gaz.,' 1856, Dec.

³ 'Brit. Med. Journ.,' 1875, i, 651.

⁴ 'Arch. der Heilkunde,' 1870, p. 23.

scrupulous cleanliness and care have not succeeded in putting a stop to them." Ample confirmation of this is afforded by the London hospitals, which, as Fergusson says, are never free from erysipelas, notwithstanding the extreme cleanliness practised in them, and despite the most abundant ventilation. In the Hôpital St. André at Bordeaux, which is highly prized as a "model institution," it appears from the statements of Pujos that even the cleanest and best constructed wards do not escape erysipelas. Ollier records that, for a long period previous to the year 1867, the severest operations had been performed at the Lyons hospital without erysipelas occurring; but from that date onwards the larger number of the cases operated upon were attacked with erysipelas, although no cause could be found for it in the condition of the place.

But the non-dependence of erysipelas upon the above-mentioned factors is proved best of all by its epidemic occurrence outside hospitals, among village populations at large, and not unfrequently in coincidence with the development or intensification of the disease in the hospitals. This fact confronts us on a large scale in the North American epidemics, in which the most remarkable circumstance was that the disease was prevalent in country districts much more frequently and much more extensively than in large and populous towns. We should not, therefore, judge of the importance of these unsanitary conditions in producing erysipelas, otherwise than we judge of their influence in other infective diseases; they afford a soil which is favorable to the development of such diseases, whether because they so affect the human organisation as to make it receptive of the proper morbid thing, or because they are concerned in some definite manner in the development of this morbid thing itself.

§ 157. THE MORBID POISON—A MICROCOCCUS.

In consideration of these facts, and in view of the circumstance that the erysipelatos process has markedly the character of an infective disease, the conviction has long

been held that the malady was caused by a specific noxious something—by a *morbid poison*. This opinion has found support in the results of inquiries made by Hüter,¹ Orth,² Nepveau,³ Lukomsky,⁴ and Klebs,⁵ and especially in the evidence recently adduced by Koch,⁶ and still better by Fehleisen,⁷ as to the constant occurrence of a species of bacterium (micrococcus), with definite characters, in the diseased tissues and in the lymphatic vessels.⁸

There need be less hesitation than ever in accepting the parasitic nature of the disease, now that its *communicability*—which had been asserted originally by English surgeons⁹ on the ground of clinical observation, and afterwards by

¹ 'Berl. klin. Wochenschr.,' 1869, 357.

² 'Arch. für experim. Pathologie,' 1873, i, 81.

³ 'Gaz. méd. de Paris,' 1873, 32.

⁴ In 'Virchow's Archiv,' 1874, lx, 418.

⁵ 'Arch. für experim. Pathol.,' 1854, iii, iv.

⁶ 'Untersuchungen über die Aetiologie der Wundinfektionskrankheiten,' Leipzig, 1878.

⁷ 'Deutsche Zeitschr. für Chirurgie,' 1882, xvi, 391, and 'Sitzungsber. der Würzb. phys.-med. Gesellsch.,' 1882, Nr. 8.

⁸ Indirect evidence of the parasitic nature of erysipelas is afforded by recent experience of the antiseptic treatment of wounds. Since the introduction of that practice erysipelas of wounds has been almost banished from surgical wards, which used to be permanent seats of the malady, and often required to be closed on account of its disastrous outbreaks. On this point, see Volkmann, 'Verhandl. der deutschen Gesellsch. für Chirurgie,' 1877, 64; Tillmanns, in the 'Deutsche Chirurgie' of Billroth and Lücke, v, 75 (for the surgical clinique of Halle); Nussbaum, 'Die chirurgische Klinik zu München im Jahre 1875,' Stuttg., 1875; and 'Leitfaden zur antiseptischen Wundbehandlung,' 3 Aufl. Stuttg. 1879.

⁹ The earliest statements occur in the reports of English hospital surgeons, such as Wells (l. c.), Dickson ('Med.-Chir. Rev.,' 1819, April, p. 615), Weatherhead ('Diagnosis between Erysipelas, Phlegmon, &c.,' Lond., 1819), Stevenson, Arnott, and others. A number of later observations on this point are collected by Volkmann (l. c., p. 155). Of still more recent date are the papers by Ollier (l. c.) for the epidemic of 1867 in the Lyons Hospital; by Borbone (l. c.) from observations in the hospital of Turin; by Erichsen for University College Hospital; by Begbie ('Edin. Monthly Journ.,' 1852, Sept., p. 243) from experiences of private practice in Edinburgh; by Tibbits ('Lancet,' 1874, June, p. 832) from hospital and private practice in Bristol; by Netten Radcliffe (l. c.) for the Oxford Infirmary; by Maclagan ('Brit. Med. Journ.,' 1876, Sept., p. 395) from private practice; by Miller ('Edin. Med. Jour.,' 1880, June, p. 1095) for the Edinburgh Infirmary; by Lücke ('Berl. klin. Wochenschr.,' 1868, p. 457) for the hospital at Bern; by Baader ('Correspondenzbl. für Schweizer Aerzte,' 1877, No. 3) for the epidemic at the village of Buus, in Neuchâtel; and by Schüller ('Deutsche Zeitschr. für Chirurgie,' 1877, viii, p. 501) for the surgical clinique at Greifswald.

French and German—has been proved with exactitude by means of the experiments on animals conducted by Orth, Koch, Tillmanns¹ and others, and most decisively of all by Fehleisen's inoculation experiments on man with micrococci of pure cultivation.² We may consider it to be an open question for the present, whence the parasite comes; also whether it reproduces itself only within the human body, or whether it grows outside as well, finding in the above-described defects of hygiene a soil specially suited for its reproduction. Again, concerning the factors which determine the type of the disease and the modifications of the same, we are unable by means of the facts known to us to come to any conclusion. In many cases it is unquestionably the individuality of the patient that makes the difference; but in many other cases, and particularly in epidemic outbreaks of the disease in its malignant form, that explanation does not apply. In these matters we are groping in the same obscurity which enshrouds the causes of mild or severe type in other infective diseases, as I have remarked more particularly when writing of scarlet fever (I, pp. 187—192).

§ 158. POINT OF ORIGIN.—MODE OF TRANSMISSION.

The opinions of observers are still divided as to the *mode of transmission of the disease*; whether an attack of erysipelas presupposes under all circumstances a breach of continuity in the surface of the body, that is to say, whether the only kind of erysipelas is an erysipelas of wounds; or whether the morbid poison may not be taken up also by the intact skin or mucous membrane. The former of these views is the one that is preferred by the larger number of experienced surgeons. The infinite difficulty, nay even the impossibility of detecting slight injuries in each particular case, especially in

¹ L. c., p. 9.

² 'Würzb. Verhandl.,' l. c. Herr Fehleisen has now (Dec., 1882) made a large number of experiments of that kind, all of them with the therapeutical intention of dispelling tumours. The whole of the experiments succeeded, and the erysipelas always ran a perfectly typical course. Through the kindness of Herr Fehleisen I have had the opportunity of seeing a successful inoculation experiment of his in Herr v. Bergmann's surgical clinique here.

the mucous membranes near where they pass into the skin, makes this question one of the most intricate in the natural history of erysipelas, and one that can never be altogether finally settled. For my own part I am constrained by force of recorded observations to give up the distinction which I drew in the first edition of this work, between simple (exanthematic) erysipelas and malignant (typhoid) erysipelas. There are many clinical observations in favour of the transmission of the morbid poison by surgical instruments, bandages or other things used by patients, that is to say, in favour of contagion in the narrower sense of the term. But in the majority of cases, no such means of transmission can be made out; and one is rather driven to think that the reproduction of the morbid poison really takes place outside the human body, and that the virus is for the most part carried by currents of air.

CHAPTER XII.

PUERPERAL FEVER.

§ 159. THE MODERN DOCTRINE STARTED BY SEMMELWEISS AND CONTINUED BY HIRSCH.

The first and most acceptable duty of modern medicine is the prevention of disease; and in practically applying that principle, medicine has achieved its most brilliant success in regard to the diseased processes incidental to wounds. Evidence of this is afforded, as we have seen, by the good effects of the antiseptic method of dressing wounds in preventing traumatic erysipelas. The prophylactic rules directed against the occurrence of hospital gangrene and the so-called septic wound-diseases have proved to be not less beneficial; and for the puerperal infective diseases, whose admission into the group of traumatic diseases hardly anyone nowadays will object to, the same holds good.

Rational prophylaxis against a disease must be based upon an acquaintance with and consideration of the causes which underlie it directly or remotely. Setting out from this principle, and working on the basis of experiences at the lying-in hospital of Vienna in 1847, Semmelweiss developed his doctrine of the prophylaxis of puerperal fever. And although his view of the causative conditions was one-sided, he was still a true pioneer in elaborating the doctrine of the origin and prevention of that disease, and his work was of great service not only to the Vienna lying-in house but to mankind at large. I take credit to myself for having, in the first edition of this work, stood forward as his exponent and for having directed the attention of the profession in Germany to his writings, which had been little noticed up to that time. My endeavour was to enlarge the doctrine of Semmelweiss

on the genesis of child-bed fever, by means of experiences of the disease collected down to a later date, or to arrive at a more comprehensive view of its etiology; and I have had the satisfaction of seeing the results of my inquiries received with approval by the first gynæcologists in Germany. I shall thus share with Semmelweiss the credit of being named a founder of the rational doctrine of the origin of puerperal fever. So much the more is it my present duty in this new edition to subject the opinions that I then expressed to a rigorous criticism in the light of knowledge gained in the interval, to turn to account for this particular infective disease the advances which science has made in the department of infective disease in general, and thus, in adherence to the facts and with as complete a presentation of them as possible, to mark the point which our knowledge of the ways of origin of puerperal fever has now reached, the point from which our practice has to start in framing the prophylactic measures to be adopted against this disastrous malady.

§ 160. PUERPERAL FEVER IN FORMER TIMES.

There is no doubt that puerperal infective disease, or—to adopt an equivalent and familiar term for the sake of brevity—puerperal fever has been prevalent in all ages, just as its geographical distribution extends over the whole habitable globe. And as the amount of this sickness varies in different parts of the world, according to the social life and hygienic circumstances of the population, there have probably been corresponding differences in the frequency of the disease in the various epochs through which the human race has passed. The extent of those differences we are certainly unable to estimate, by reason of the scanty notices of the disease which have come down to us from ancient and mediæval times, and the absence of accurate information from the sixteenth and seventeenth centuries.

In the epidemiological writings of Hippocrates,¹ there occur a number of excellently drawn up clinical histories of puerperal fever, and in the

¹ 'Lib. Epidemiorum,' i, sect. iii, Aegra Nr. 4, 5, 11, ed. Littré ii, 690, 694, 708; 'Epid.,' lib. iii, sect. ii, Aegra 10, 11, 12, e. c. iii, 60—62; lib. iii, sect. iii, Aegra 2, 14, e. c. iii, 108, 140.

pseudo-Hippocratic treatise 'De morbis mulierum' the subject is considered at length. Moreover it follows from the account² given by Hippocrates of the diseases among the inhabitants of Thasos that the malady had been common in those days and had occurred in a sort of epidemic. ("γυναῖκες δὲ ἐνόσησαν μὲν πολλαί . . . ἐδυστόκειον δὲ πλείσται, καὶ μετὰ τοὺς τόκους ἐπενόσκειον, καὶ ἔθνησκον αὐταὶ μάλιστα.") Celsus,³ however, confines himself to one remark, which probably points to puerperal fever ("mulier ex partu si cum febre, vehementibus etiam et assiduis capitis doloribus premitur, in periculo mortis est"); and Galen⁴ contributes nothing of his own, but merely repeats the statements of Hippocrates. Soranus and Moschion, the gynæcologists of the Greek medicine, make no mention whatsoever of the disease; and Arabian physicians such as Avicenna,⁵ as well as the physicians of the middle ages, have nothing more to say than that women in childbed sometimes took fever, which led to their death.

In the medical writers of the sixteenth and seventeenth centuries, such as Trincavella,⁶ Mercado,⁷ Rivière,⁸ de la Boë,⁹ Sennert¹⁰ and Sydenham,¹¹ we find statements about puerperal fever which go rather more into particulars, although they hardly do justice to the properly clinical side of the question. Willis was the first to appreciate correctly the peculiar character of the disease as compared with other febrile processes; he introduced the name "febris puerperarum," and undertook a searching inquiry into the beginnings and course of child-bed fever; he even went so far as to connect the origin of the disease with laceration of the uterus during labour and to refer it to a "miasma venenatum."¹² After Willis came Strother,¹³ Hoffmann,¹⁴ and others.

Although, as we have said, it is impossible to estimate from the professional writings at our service, the extent to which child-bed fever had been prevalent in past times; yet we are justified by the fact, observed during the present

¹ Lib. i, § 49—54, e. c. viii, 108, *seq.*

² Lib. i, sect. ii, § 8, e. c. ii, 646.

³ 'De med.,' lib. ii, cap. 8.

⁴ In the commentaries to the above-quoted passages in Hippocrates.

⁵ 'Canon,' lib. iii, Fen. xxi, tract. ii, cap. 33, Venet., 1564, i, 929.

⁶ 'Dé ratione curandi, &c.,' lib. xi, cap. ii, ff. in 'Opp.,' Lugd., 1586, i, 291, *seq.*

⁷ 'De mulierum affect.,' lib. iv, cap. x, xi.

⁸ 'Praxis med.,' lib. ix, Norimb., 1688, 287.

⁹ 'Prax. med.,' lib. iii, cap. viii, § 13, *seq.*, Amstel., 1679, 554.

¹⁰ 'Pract. med.,' lib. iv, sect. vii, cap. xi, Wittenbg., 1660, 467.

¹¹ 'Diss. epistol. ad Cole de affect. hyster.,' in 'Opp.,' Genev., 1736, i, 279.

¹² 'De febribus,' cap. xvi, in 'Opp.,' Amstel., 1682, 124.

¹³ 'Critical Essay on Fevers,' cap. ix, Lond., 1718, 212.

¹⁴ 'Med. rationalis system,' tom. iv, Pars i, sect. ii, cap. x, in 'Opp.,' Genev., 1748, ii, 156.

century and the preceding, that it clings mostly to lying-in hospitals, in believing that the important place which puerperal fever now takes in the statistics of sickness and mortality of civilised countries dates no farther back than the end of the seventeenth or beginning of the eighteenth century, or from the period when the first maternity hospitals or other institutions for the reception of the lying-in were established.

§ 161. PRESENT DISTRIBUTION.

Regarding the *geographical distribution of puerperal fever*, the want of information from many parts of the world, particularly those within tropical and subtropical latitudes, does not permit us to do more than conclude generally that the disease extends over the whole habitable globe. For the same reason I am unable to say whether any considerable differences exist in the number of cases at various parts of the world, not including the cases of child-bed fever in lying-in hospitals. As a general rule, child-bed fever would appear to occur most frequently in highly-civilised countries with a crowded population and situated in higher latitudes. Without attaching too much importance to the following statistics of the disease collected from some of the *civilised States of Europe*, I think they may be used to form an approximate estimate.

Table of Deaths from Puerperal Fever per 1000 Deliveries.¹

Place.	Period.	Deaths per 1000.	Authority.
St. Petersburg ¹	1845-59	7.0	Hugenberger. ²
Sweden	1861-75	5.7	Netzel. ³
Norway ⁴	1859-68	5.7	Vogt. ⁵
Denmark	1866-74	6.0	Weis. ⁶
England.....	1818-41	8.0	} d'Espine. ⁷
England.....	1847-50	6.0	
Prussia	1816-75	8.0	Boehr. ⁸
Baden.....	1851-63	7.6	Hegar. ⁹
Belgium.....	1851-55	6.0	d'Espine. ⁷
Geneva	13 years	8.0	d'Espine. ⁷
Genoa.....	1857-66	4.2	Sormani. ¹⁰

¹ For footnotes see bottom of next page.

For *Turkey*, and for Constantinople in particular, Rigler¹ says that, although the absence of lying-in hospitals makes child-bed fever rarer than in most other cities of Europe, yet the disease does occur from time to time. Also the most northern countries of Europe are by no means exempt from it. In *Iceland* it is not so very rarely met with,² cases occurring from time to time even in large numbers, as in 1842 and 1845. The same holds good for *Greenland*, where groups of puerperal fever cases occur, at all events now and then, as in 1844 and 1847.³ In the cultivated parts of *North America* it is just as extensive as on European soil. It is of interest to notice the remark of a United States authority,⁴ on the occurrence of puerperal fever in that country, in a criticism of the gynæcological work of Lee (1835): "In this country we have fortunately had but little experience of the alarmingly fatal epidemics that have spread their devastating influence over different sections of Great Britain;" and Hildreth,⁵ writing from Washington, Ohio, in 1830, says, "Puerperal fevers are much less common than in more populous places." From *Central America* and the *West Indies* our information about child-bed fever is meagre, but it serves to prove that the disease is no stranger to these countries. For the Continent of Africa, I have found only one notice, by Pruner,⁶ according to which puerperal fever in Egypt is neither so common nor so general as in Europe,

¹ 'Die Türkei und deren Bewohner, &c.,' Wien, 1850, ii, 338.

² Sehleisner, 'Island undersøgt, &c.,' Kjöbenh., 1849, 50.

³ Account in 'Sundhedskoll. Forhdl. for Aaret 1844,' 57; Lange, 'Bemaerkn. om Grönlands Sygdomsforhold.,' Kjöbenh., 1864, 40.

⁴ 'Amer. Journ. of Med. Sc.,' 1835, Feb., 439.

⁵ *Ib.*, 1830, Feb., 300.

⁶ 'Krankheiten des Orients,' Erlang., 1847, 278.

¹ The death-rate among women delivered in private houses.

² See the list of authors at the end of the chapter.

³ 'Hygiea,' 1879, xli, 177.

⁴ Including those who died in Maternity hospitals.

⁵ 'Norsk. Mag. for Laegevidensk.,' 1872, iii, Raekke ii, 414.

⁶ 'Hygien. Meddelelser,' 1875, Nye R., i, 124.

⁷ 'Gaz. des hôpit.,' 1858, Nr. 54, 213.

⁸ 'Zeitschr. für Geburtsh. und Gynäkol.,' iii, 81.

⁹ 'Die Sterblichkeit während der Schwangerschaft, &c.,' Freiburg, 1868, 25.

¹⁰ 'Geogr. nosol. dell' Italia,' Roma, 1881, 176.

although it has sometimes been met with to a more considerable extent, as, for example, in 1844, when a "puerperal influence" is said to have reached from Alexandria to the Thebaid. In Port Said, as Vauvray¹ tells us, it is almost unknown. In *India*, according to Webb² and Twining,³ child-bed fever is by no means rare among the Hindu women, and it is sometimes prevalent to a considerable extent. The observations of Baelz⁴ leave no doubt of the occurrence of the disease in Japan, even if it be only in isolated cases. On the *Australian Continent*⁵ and in *New Zealand*⁶ puerperal fever would appear to have been very rare down to about the middle of the present century; but I have no more recent information of the disease in those colonies. In *Tasmania* it was first seen, so far as Dempster⁷ knew, in the autumn of 1833 in the district of Norfolk, several women having taken it together. According to later accounts by Hall⁸ and Miller,⁹ child-bed fever is far from uncommon there, whether in the lying-in hospital (where it was epidemic in 1851-52) or among women confined in their own homes. The only notice for *South America* that I know of is one by Feris,¹⁰ according to which the disease was disastrously prevalent at Monte Video in 1875.

§ 162. EPIDEMIC OUTBREAKS.

Although our information on the geographical distribution of puerperal fever is very incomplete, and the statistical returns of the disease among the populations of the various parts of the world are below the real total; yet

¹ 'Arch. de méd. nav.,' 1873, Sept., 188.

² 'Pathologia Indica,' Lond., 1848, 336.

³ 'Clinical Illustrations of the more important Diseases of Bengal,' Calcutta, 1835, ii, 433.

⁴ 'Infectionskrankheiten in Japan,' Yokohama, 1882, 5.

⁵ Account in 'Lancet,' 1845, Sept., 321.

⁶ Thomson, 'Brit. and Foreign Med.-Chir. Rev.,' 1854, Oct.

⁷ 'Transact. of the Calcutta Med. Soc.,' 1836, vii, 358.

⁸ 'Transact. of the Epidemiol. Soc.,' 1866, ii, 69.

⁹ 'Glasgow Med. Journ.,' 1878, Aug., 345.

¹⁰ 'Arch. de méd. nav.,' 1879, Oct., 253.

it is an undoubted fact that the disease has been infinitely more common, whether in *sporadic* cases, or in *epidemics* and *endemics*, within maternity hospitals or in the lying-in departments of general hospitals than outside them, that is to say, than among women confined at their own dwellings. At the same time it is not altogether rare for child-bed fever to be prevalent to a very considerable extent among the latter; and sometimes it even takes the character of an epidemic.

In the following table I have put together, in chronological order, a list of the epidemics of puerperal fever that have come to my knowledge. Although I am far from claiming it to be a complete list of all the observations on record, yet it will suffice to bring out several points of interest for the history of the disease and for the study of its etiology.

Table of Epidemics of Puerperal Fever.

Time.		Place.	Area of diffusion.	Authority.
1664	...	Paris	Obstetric wards of the Hôtel Dieu.....	Peu. ¹
1672	...	Copenhagen.	In the town ²	Bartholin.
1713	...	Rouen, Caën.	Ditto	de la Motte.
1723	...	Frankft. on M.	Ditto	Hoffmann.
1736-37	Winter	Paris	Ditto	Foderé.
1746	Jan.—March	Paris	Hôtel Dieu and in the town.....	Malouin.
1750	Spring	Lyons	Hôtel Dieu.....	Pouteau.
1760	July—Dec.	London	British Lying-in Hosp.	Leake.
1761	...	Aberdeen ...	?	Mackintosh.
„	May—July	London	In a small private Lying-in Hosp.	White.
1765-66	...	Copenhagen.	Lying-in Hosp.	Saxtorph.
„	...	Rotterdam...	In the town	Bikker.
„	...	Derbyshire...	In various parts of the county in this and following years	Butter.
1767	...	Gröningen...	Vicinity of the town...	v. Döveren.
„	...	Heugon	In the parish	Lepecq de la Cloture.
„	Dec.—May	(Normandy) Dublin	Rotunda	Jos. Clarke.

¹ The authorities named in this column are given in alphabetical order at the end of the chapter.

² “In the town” means that the disease occurred outside the lying-in hospital.

	Time.	Place.	Area of diffusion.	Authority.
1769-70	Nov.—May	London	Westminster, British, and other Lying-in Hosps.; also in the town	} Leake, White.
1770	Autumn	Vienna	St. Marx Lying-in Hosp.	
"	...	Rotterdam...	In the town	Fauken.
1771	...	London	In several Lying-in Hosps.	Bikker.
1772	Feb. and fol. months	Edinburgh...	Obstetric wards of the Infirmary	} Young, Clarke.
1774-75	Winter	Paris	Hôtel Dieu, and many cases in the town ...	
1774	March—May	Dublin	Rotunda	Ref. I.
1777	...	Stockholm...	Lying-in Hosp. ¹	Jos. Clarke.
"	Summer	Vienna	Obstetric wards of the General Hosp.	Netzel.
1778	...	Copenhagen.	Lying-in Hosp.; also a few cases in the town	Stoll.
"	Summer	Berlin	Cases in the town....	Saxtorph.
"	Jan.—March	Paris	Hôtel Dieu; also a few cases in the town ...	Selle.
1780	February	Berlin	Cases in the town....	Geoffroy.
1781	Sept.—Nov.	Cassel	Lying-in Hosp.	Selle.
1781-82	Nov.—Jan.	Paris	Hôpital Vaugirard ...	Osiander.
1782	Jan. and fol. months	Copenhagen.	Lying-in Hosp.; also cases in the town ...	Doublet.
1783-84	Dec.—March	Gladenbach (Giessen)	Many cases in town and vicinity	Tode.
1786	Sept.—Dec.	Copenhagen.	Lying-in Hosp.	Diel.
1786-87	Sept.—July	Arzago (Lombardy)	Widely prevalent in the village	Bang, Salomonsen.
1787	Spring and Summer	Poitiers	Many cases in the town	Cerri.
"	Mar.—April	Dublin	Rotunda	Lamarque.
1787-88	July—Jan.	London	In a Lying-in Hosp.; also cases in the town	Jos. Clarke.
1788-89	Nov.—Jan.	Dublin	Rotunda	John Clarke.
1789-92	Dec.—Oct.	Aberdeen ...	Lying-in Hosp.; also many cases in the new town	Jos. Clarke.
1791-92	Oct.—April	Copenhagen.	Lying-in Hosp.; a few cases in the town ...	Gordon.
1792-93	Dec.—May	Vienna	Lying-in Hosp.	} Boysen Rinck.
1793	...	Stockholm...	
"	...	Amsterdam .	Lying-in Hosp.	Ficker.
1793	...	Rouen	Hospice de L'humanité	Netzel.
				Thijssen.
				Leroy.

¹ In using Netzel's Reports on Child-bed Fever in the Lying-in Hospital of Stockholm from 1755 to 1877, I have taken all those years in which the mortality was more than 6 per cent. of the women confined.

Time.		Place.	Area of diffusion.	Authority.
1794	...	Stockholm	Netzel.
1795-96	Autumn and Winter	Vienna	Lying-in Hosp.	Jaeger, Nebel.
1798	Mar.—April	Créteil (Dpt. Seine)	Many cases in the village	Ref. II.
1799 } 1800 }	Winter	Grenoble ...	Many cases in the town	Ref. III.
1801-2	Dec.—Feb.	Trier	Many cases in town and vicinity	
1803	...	Dublin	Rotunda	Burekhardt.
1805	Aug.—Sept.	Rostock	Several cases in town and vicinity	Douglas.
1808-12	...	Yorkshire ..	At Barnsley, Leeds, Huddersfield, and other towns	Nolde.
1810	Jan.—May	Milan	Santa Catarina Lying-in Hosp.	} Hey, Bradley.
1810-11	Winter	Landsberg..	Several cases in the town and vicinity ..	
"	"	Dublin	Rotunda	Punch.
1811	...	Stockholm ...	Lying-in Hosp.	Douglas.
"	Summer and Autumn	London	Many cases in the vicinity	Netzel.
1811-12	June—April	Heidelberg..	Lying-in Hosp.; occasional cases in town	} Ramsbotham Nägele, Bayrhofer.
1812	Jan. and fol. months	London	Many cases at Holloway & other suburbs	
"	...	Durham	Numerous cases at various localities in the county	Dunn.
1812-13	Winter	Dublin	Rotunda; occasional cases in the town ...	} Armstrong. Douglas, Brenan.
1813	Jan.—Dec.	Northumberland	At Sunderland, Alnwick, Newcastle, and other places	
1813-14	July—June	Abingdon ... (Berkshire)	In the town and neighbourhood	Armstrong.
1814	Spring	Prague	Lying-in Hosp.	West. Quadrat.
1814-15	Winter	Edinburgh..	Maternity; a few cases in the town	
1815	...	Dublin	Rotunda	Cardiff.
1816-17	...	Philadelphia	Pennsylvania Hosp. ...	Hodge.
1817	Summer	Würzburg ..	Lying-in Hosp.	d'Outrepoint, I.
"	...	Stockholm ...	Ditto	Netzel.
1818	Autumn	London	In a Lying-in Hosp., and some cases in the town	Armstrong. Cliet.
"	Sept.—Dec.	Lyons	Lying-in Hosp.	
1818-19	Oct.—Aug.	Prague	Ditto, and many cases in the town	Bischoff.
"	Oct.—March	Würzburg ..	Lying-in Hosp.	d'Outrepoint, I, II.

Time.		Place.	Area of diffusion.	Authority.
1819	...	Stockholm ...	Lying-in Hosp.	Netzel.
"	May—July	Lyons	Ditto	Cllet.
"	July—Nov.	Vienna	Ditto, and a few cases in the town and sub- urbs	Ref. IV.
"	...	Glasgow	Many cases in certain suburbs	Burns.
1819-20	Dec.—March	Würzburg ...	Lying-in Hosp.	d'Outrepont, I-III, Schloss.
"	Winter	Stockholm ...	Ditto	Cäderschjöld, I.
"	"	Kiel	Ditto	Michaelis.
"	Dec.—Aug.	Dresden	Ditto	Carus, I.
"	Oct.—Jan.	Bavaria	At Bamberg, in the Lying-in Hosp., and subsequently many cases in the town; numerous cases at Ansbach, Nürnberg, and Dillingen	} Pfeufer, Schilling.
"	Winter	Dublin	In the Lying-in Hosp.	
1821	Spring and Summer	Lyons	Charité	Douglas, Collins. Baudelocque.
1821-22	March 21— Sept. 22	Scotland	Many cases at Edin- burgh, Glasgow, Stir- ling and other places	} Campbell, Mackintosh
1822-23	Winter	Marburg	Lying-in Hosp.	
"	"	Vienna	Ditto	Busch. Lippich.
1823	January	London	Queen Charlotte's Ly- ing-in Hosp.	Ferguson.
"	...	Dublin	Rotunda	Collins.
1824	Winter	London	In a Lying-in Hosp. ...	Gooch.
"	Jan.—May	Dresden	Lying-in Hosp.	Carus, II.
1824-25	Nov.—Jan.	Munich	Ditto	Graf.
1825	Jan.—April	Berlin	Ditto, and many cases in the town	Siebold.
"	...	London	In a Lying-in Hosp. ...	Ferguson.
"	...	Hanover	Lying-in Hosp.	Dommes.
"	...	Prague	Ditto	Quadrat.
1825-26	Winter	Edinburgh ..	Maternity Hosp.	Sidey.
"	Nov. and fol. months	Stockholm ...	Lying-in Hosp.	Cäderschjöld, II.
1826	...	Nastätten ... (Nassau)	Widely spread in the locality	Ricker.
"	...	Stockholm ...	Lying-in Hosp.	Netzel.
"	Jan.—June	Berlin	Charité	Neumann.
"	...	Dublin	Rotunda	Collins.
"	...	Paris	Hôtel Dieu	Baudelocque.
"	...	Birmingham	Lying-in Hosp.; a few cases in the town ...	Ingleby.

Time.		Place.	Area of diffusion.	Authority.
1827	April—May	Barmen	Many cases in the town	Sonderland.
1827-28	Nov.—Jan.	Neuenhaus... (Bentheim)	Many cases in town and vicinity	Miquel.
1828	...	Dublin	Rotunda	Collins.
1828-29	Autumn and Winter	London	Westminster Lying-in Hosp.; many cases in the town.....	} Hingeston, } Gooch, } Ferguson.
"	...	Amsterdam	Lying-in Hosp.	
1829	...	Vienna	Ditto	Tilanus.
"	...	Hanover.....	Ditto	Dommes.
"	...	Dublin	Rotunda	Collins.
"	Feb.—Aug.	Copenhagen.	Lying-in Hosp.	Kayser.
"	Whole year	Paris	La Maternité	Tonnellé, Duplay.
1830	Winter	Toulouse.....	Many cases in the town	Brun.
"	...	Prague	Lying-in Hosp.	Quadrat.
"	April—June	Dresden	Ditto	Haase, I.
"	...	Kiel.....	Ditto	Michaelis.
1830-31	Summer of 1830 to Autm. of 1831	Giessen	Ditto	Ritgen.
"	Dec.—March	Paris	La Maternité.....	Cruveilhier Nonat.
"	Winter and Spring	Manchester	Lying-in Hosp.; many cases in the town ...	Robertson.
"	...	Philadelphia	Pennsylvania Hosp...	Hodge.
1831	...	Usingen.....	Many cases in the village	Ricker.
"	...	Stockholm ...	Lying-in Hosp.	Netzel.
"	Autumn	Aylesbury ...	Many cases in the town	Ceely.
"	"	Plymouth ...	Ditto	Blackmore.
"	Aug.—Dec.	Paris	Hôtel Dieu and Mater- nité	Nonat.
1832	Winter	Munich	Lying-in Hosp.	Martin, I.
"	April—June	Bonn	Many cases in Lying- in Hosp. and in the town.....	Levin.
1833	...	Usingen.....	Many cases in village (2nd time)	Ricker.
"	...	Vienna	Lying-in Hosp.	} Ingleby, } Elkington.
"	Aug.—Sept.	Birmingham	Ditto; some cases in the town	
"	Autumn	Edinburgh...	Numerous cases in the town.....	Patterson.
"	Feb.—March	Philadelphia	Pennsylvania Hosp...	Hodge.
1833-35	Oct., 1833— May, 1835	Prague	Lying-in Hosp.	Quadrat.
1834	...	Gratz	Ditto	Schöller.
"	Jan.—May	Vienna	Ditto	Bartsch, Martin, II.

Time.		Place.	Area of diffusion.	Authority.
1834	Autumn	Dublin	New Rotunda, following a few cases in other lying-in houses and in the town.....	Beatty, I.
"	...	Paris	La Maternité.....	Tanchou.
"	Jan.—March	Trarbach ... (Coblenz)	Many cases in the town	Graff.
"	...	Bamberg ...	Lying-in Hosp., and in the town	Rapp.
1834-35	Sept.—Mar.	Kiel.....	Ditto, ditto.....	Michaelis.
1835	...	Paimbœuf ... (Loire infér.)	Many cases in the arrondissement	Aubinais.
"	Mar.—April	Hanover.....	Lying-in Hosp.	Dommes.
"	Autumn	Würzburg ...	Ditto, and in the town	d'Outrepont, IV.
1835-36	Winter	Kiel.....	Lying-in Hosp.	Michaelis.
1836	Spring	Hadamar ... (Nassau)	Small epidemic in the place	Ricker.
"	Winter and Spring	Vienna	Lying-in Hosp.	Hauner, Helm.
"	Jan.—Feb.	Dublin ... }	New Rotunda	Beatty, II.
1837	January	Dublin ... }		
"	Feb.—May	Dresden	Lying-in Hosp.	Haase, II.
"	March—May	Copenhagen.	Ditto; also some cases in the town.....	Müller, Ref. V.
1837-38	Dec.—April	Greifswald...	Ditto, ditto.....	Berndt.
1838	Jan.—May	London	In a Lying-in Hosp. ...	Ferguson.
"	Autumn	Dresden	Lying-in Hosp.	Haase, II.
"	Mar.—Aug.	Paris	Clinique	Dubois, I, Voillemier.
1838-39	Nov.—July	Prague	Lying-in Hosp.	Jungmann, I.
1839	Jan.—March	Dresden	Ditto	Haase, III.
"	May—July	Wangen	All the women confined during that period	Zengerle.
1839-40	Oct.—Jan.	Copenhagen.	Lying-in Hosp.	Kayser, Müller.
1840	Oct.—Dec.	Dülmen	Many cases in the parish	Ref. VI.
"	Nov.—Dec.	Stockholm...	Lying-in Hosp.	Elliot, Netzel.
"	...	Paris	Hôtel Dieu.....	Bourdon.
"	Spring	Prague	Lying-in Hosp.	Jungmann, II.
1840-41	Sept.—Mar.	Berlin.....	Charité; also many cases in the town ...	Schönlein, Jonas, de Lingen, Diemer.
"	Dec.—Mar.	Halle	Lying-in Hosp.	Litzmann.
1841	...	Stockholm...	Ditto	Netzel.
"	Jan.—March	Doncaster ...	Many cases in the town	Storrs.
"	March	Paris	Maternité and Clinique	Dubois, II, Ref. VII.

	Time.	Place.	Area of diffusion.	Authority.
1841-42	Dec.—May	Millersburgh (Ohio)	Lying-in House; afterwards many cases in the town	Bowen.
1842	Jan.—March	Peitz	Numerous cases in the town.....	Schlesier.
„	Mar.—April	Philadelphia	Lying-in Hosp.....	Wilson.
„	Feb.—May	Paris	La Maternité.....	Ref. VIII.
„	Jan.—Dec.	Gratz	Lying-in Hosp.....	Götz.
„	Feb.—June	Rennes	Hôtel Dieu.....	Botrel.
1843	...	Rouen.....	Lying-in Hosp.....	Hervieux.
„	Spring and Summer	Dorpat	Ditto; also several cases in the town ...	Koch.
„	Jan.—April	Paris	In all the Lying-in Hospitals, and many cases in the town as well	Bouchut, Bidault.
„	Aug.—Dec.	Paris		
1844	...	Stockholm...	Lying-in Hosp.....	Netzel.
„	...	Rouen.....	Ditto	Hervieux.
„	Sept.—Nov.	Paris	Ditto	Bouchut, Bidault.
„	Spring	Rennes	Hôtel Dieu.....	Botrel.
„	July—Oct.	Girresheim .. (near Düsseldorf)	In the village.....	Scheider.
„	Sept.—Nov.	Copenhagen.	Lying-in Hosp.....	Kayser, Ref. IX.
„	Feb.—June	Hads-Herred (Aarhuus, Jutland)	Throughout the parish	Schäffer, Jespersen.
„	Nov.—Dec.	Aalborg	Many cases in the town	Speyer.
„	Summer	Jacobshavn .. (N. Greenland)	Small epidemic in the settlement	Kayser, II.
1844-45	Winter	Lyons	Lying-in Hosp.....	Vernay.
1845	March	Dublin	Rotunda	McClintock, I
„	...	Rouen.....	Lying-in Hosp.....	Hervieux.
1845-46	Winter	Paris	Chiefly in Hôtel Dieu and Charité; also in Hôpital St. Louis and La Pitié, and many cases in the town ...	Ref. XII.
„	„	Lyons	Lying-in Hosp.....	Vernay.
1846	...	Stockholm...	Ditto	Netzel.
„	...	Vienna	Ditto	
„	...	Rouen.....	Ditto	
„	Spring	St. Petersburg.	Obstetric Institute ..	Hugenberger
„	July	Gröningen...	Lying-in Hosp.....	Baart de la Faille.
1846-47	Nov.—Feb.	Würzburg ...	Ditto	Heymer.
„	Dec. and fol. months	Toulouse.....	Ditto, and in Hôtel Dieu.....	d'Orbeastle.
„	Dec.—March	Berlin	Charité	Virchow, I.

Time.		Place.	Area of diffusion.	Authority.
1847	April	Stuttgart ...	Lying-in Hosp.	Elsässer, I.
"	...	Ohio	Numerous cases in many parts of the State	Holston.
1848	...	Kiel.....	Lying-in Hosp.	Michaelis.
"	Spring and Autumn	St.Petersbrg.	Obstetric Institute ...	Hugenberger
"	December	Bornholm ...	Many cases throughout the population...	Ref. X.
1849	...	Stockholm ...	Lying-in Hosp.	Netzel.
"	...	Bern	Ditto	Herrmann.
1849-50	Sept.—June	Stuttgart ...	Ditto; also many cases in the town, and in other parts of Würtemberg	Elsässer, II.
1849-50	...	Tübingen ...	Lying-in Hosp.	Reuss.
1850	March	Copenhagen ...	Many cases in the town	Hassing.
"	...	Stockholm ...	Lying-in Hosp.	Netzel.
"	Spring	Rezé	In the village.....	Galicier.
		(Loire infér.)		
1850-51	Nov.—Feb.	New York ...	Maternity attached to the Coloured Home Hosp.	Parkins.
1851	Oct. and fol. months	Stockholm ...	Gen. Lying-in Hosp...	Retzius, I.
"	...	Christiania .	Ditto; also a few cases in the town.....	Faye.
"	July—Sept.	Bordeaux ...	Many cases in the town	Burguet.
1851-52	...	Pennsylvania	Numerous cases in many parts of the State	} Leasure, Ref. XI.
"	Sept.—Jan.	Brakel	Many cases in the town	
"	...	(Minden)		Disse.
"	...	Tasmania ...	Lying-in Hosp.	Hall.
"	Dec.—May	Leer	In the village and country around	Kirchhoff.
1852	...	Paris	Clinique	Dubois, III.
"	Spring	St.Petersbrg.	} Obstetric Institute .	Hugenberger
1852-53	Winter	St.Petersbrg.		
1853	Feb. and fol. months	Gröningen...	Lying-in Hosp.	Baart de la Faille.
"	Winter	Berlin	Obstet. division of the Charité	Credé.
1854	Autumn	Paris	La Maternité	Charrier.
"	Feb.—April	Munich	Many cases in the town	Herliner.
"	July—Sept.	St.Petersbrg.	Obstetric Institute ...	Hugenberger
1854-55	June—May	Dunkirk.....	Many cases in the town	Zandyk.
"	Dec.—Feb.	Dublin	Rotunda; also some cases in the town ...	} McClintock, II.
1855-56	Nov.—May	St.Petersbrg.	Obstetric Institute ...	
1856	Feb.—March	Middleburg .	In the town, and at other places in Zealand	Doornick.

	Time.	Place.	Area of diffusion.	Authority.
1856	...	Paris	Clinique	Dubois, III.
1856-57	Dec.—June	Munich	New Lying-in Hosp.; subsequently in other Lying-in Hosp., and many cases in town and vicinity	Martin, III.
„	Nov.—March	Strasburg ...	Obstetric Clinique; also cases in town and vicinity	Levy.
1857	Mar.—April	Prague	Lying-in Hosp.	Dor.
1857-58	Winter	Berlin	Charité	Virchow, II.
„	Whole year	New York ..	Bellevue Hosp.	Barker.
1858	March—May	Prague	Lying-in Hosp.	Heiss.
„	Autumn and Winter	Berlin	Obstetrical Clinique...	Martin.
„	June—Dec.	Helsingfors .	Lying-in Hosp.; afterwards many cases in the town	} Pipping-skjöld.
„	May—June	Trient	Alle Laste Institution	
„	...	Bern	Lying-in Hosp.	Hermann.
„	...	Paris	Clinique	Dubois, III.
1858-59	Nov.—Mar.	St. Petersburg.	Obstetric Institute ...	Hugenberger Grünewald.
1859	Feb.—April	Würzburg ..	Lying-in Hosp.; also cases in the town and vicinity	} v. Franque, I, Scanzoni.
„	July—Nov.	St. Petersburg.	Obstetric Institute ...	
„	February	Amsterdam .	Maternity wards of the Hosp.	Hugenberger Lehmann.
1859-60	...	Prague	Lying-in Hosp.	Weber.
„	Winter	Berlin	Charité; Royal Maternity Institution; also many cases in the town	} Martin, IV, Nagel.
„	...	Stockholm ...	Lying-in Hosp.	
„	Aug.—Jan.	Giessen	Ditto	Retzius, II.
1860	Jan.—June	Würzburg ..	Ditto	Kehrer.
„	...	Munich	Ditto	v. Franque, II.
1860-61	Winter	Strasburg ...	Ditto	Hecker, I.
1861	Jan.—Sept.	Prague	Ditto; and many cases in the town also	Sieffermann.
„	Jan.—Feb.	Paris	Hôpital St. Louis	Löschner.
1861-62	Winter	Dublin	Rotunda; also a few cases in the town and vicinity	Pihan-Dufeillay.
„	Oct.—Aug.	Munich	Lying-in Hosp.	Denham.
„	Winter	Vienna	Ditto	Hecker, II.
1862-63	...	Jena	Ditto	Späth.
1863	...	Stockholm ...	Ditto	Rupert.
1863-64	Oct.—Feb.	Olmütz	Ditto	Netzel.
				Schoefl.

Time.		Place.	Area of diffusion.	Authority.
1863-65	...	Schwezingen	Numerous cases in the town and four adjoining villages	Naumann.
1864	...	} Stockholm ...	Lying-in Hosp.	Netzel.
1865	...			
"	Spring	Mannheim ...	Slight epidemic in the town	Stehberger.
1866	June—Aug.	Lyons	Maternity department of the Charité	Fonteret.
"	May	Dürkheim ...	Slight epidemic in the town	Kaufmann.
1868	...	Lyons	Hôpital de la Croix-Rousse	Guyenot.
1868-69	Winter	Montpellier .	Lying-in Hosp.	Serre.
"	"	Berlin	Obstetrical wards of the Charité	Schultze.
"	"	Paris	Hôpital de la Pitié ...	Ref. XIII.
1869	Autumn	Paris	Hôpital St. Antoine ...	Lorain, V. Martin, V. Spiegelberg.
1870	January	Breslau	Lying-in Hosp.	Parry.
1870-74	...	Philadelphia	Severe epidemic in the Philadelphia Hosp. .	Netzel.
1872	...	Stockholm ...	Lying-in Hosp.	Kraus.
"	Oct.—Nov.	Bensheim ... (Hesse)	In the village	
"	Feb.—April	Paris	Hôpital St. Antoine ...	Quinquaud.
1872-73	Nov.—Jan.	Leipzig	Slight epidemic in the town	Ahlfeld.
1873	Winter	Melbourne ...	Lying-in Hosp.; also in the town	Ref. XIV.
1873-74	Winter and Spring	New York ...	Matern. departments of the Bellevue and Charity Hospitals...	Lusk.
1878	Jan.—May	Paris	Hôpital Beaujon	Chevance.
1879	December	Cracow	Obstetrical Clinique...	Mars.
"	...	Schwen- } ingen... }	Slight epidemic in the village	Haehnle.
"	Mar.—Aug.	Berlin	Obstetrical wards of the Charité	Runge.

§ 163. EPIDEMICS MOST FREQUENT IN COLD AND WET WEATHER.

Although we may assume without hesitation that child-bed fever is more common in tropical and subtropical regions than the scanty information from such countries would lead us to suppose, the disease is at any rate rarer in them than in higher latitudes. Without doubt this is explained in great part by the fact that lying-in institutions, which are the chief seats of the malady, have been introduced to a very much smaller extent in the former than in the latter, being in fact non-existent in the more uncivilised countries. But the relative exemption of warm countries is to be explained in part also by the *climate*. I find evidence of this in the fact that in the temperate and high latitudes the maximum of sickness falls in the *cold season* and the minimum in the warm. Almost all the observers, both old and new, such as Rivière, Willis, Bartholin, Manning,¹ de la Roche,² Duges,³ Conquest,⁴ Dubreilh,⁵ Virchow,⁶ and Hervieux,⁷ are agreed in saying that the epidemic or endemic prevalence of the disease falls in winter or spring; and these observations of individual authorities are fully borne out by statistics.

Of 195 epidemics in the above table, for which the time of prevalence is accurately given, the numbers in the several seasons are as follows:

Winter	66	Autumn and Winter . .	11
Spring	34	Summer	10
Winter and Spring .	25	Summer and Autumn .	7
Autumn	21	Spring and Summer .	5

Sixteen epidemics lasted through more than one season. According to Hugenberger's⁸ data for the Obstetric Institute of St. Petersburg, the following are the proportions for the several seasons during a period of fifteen years:

¹ 'Treatise on Female Diseases,' Lond., 1771, 360.

² 'Recherches sur la nature et le traitement de la fièvre puerpérale, &c.,' Par., 1783.

³ 'Journ. gén. de méd.,' 1828, tome cv, 98.

⁴ 'Observations on Puerperal Inflammation, &c.,' Lond., 1830.

⁵ 'De la fièvre puerp. épidémique,' Bordeaux, 1848.

⁶ 'Monatsschr. für Geburtskd.,' 1858, xi.

⁷ L. c. (see list of authors), p. 58.

⁸ 'Das Puerperalfieber im St. Petersburger Hebammen-Institute, &c.,' St. Petersburg, 1862, 5.

Table of Sickness and Mortality from Puerperal Fever at the St. Petersburg Lying-in Hospital for the several seasons during 15 years.

	Confinements.	Cases of puerperal fever.	Deaths from puerperal fever.
Winter	2106	405 or 19'23%	88 or 4'18%
Spring	1934	292 or 15'09	66 or 3'41
Autumn	2069	310 or 14'98	45 or 2'17
Summer	1927	227 or 11'77½	39 or 2'02

The following are Späth's¹ figures for the two clinics of the Allgemeine Gebärhäus of Vienna during a period of twenty-four years (1840—1863):

Similar Table for the Vienna Allgemeine Gebärhäus, 1840—1863.

	First clinique.			Second clinique.		
	Confinements.	Deaths.	Per cent.	Confinements.	Deaths.	Per cent.
January	8307	535	6'4	6776	243	3'5
February ...	7926	420	5'2	6542	180	2'7
March	8323	457	5'4	7068	289	4'0
April	7688	413	5'3	6520	237	3'6
May	8239	302	3'6	6834	183	2'6
June	7149	221	3'0	6230	142	2'2
July	6900	271	3'9	6071	125	2'0
August	6699	260	3'8	6067	126	2'0
September ..	6976	242	3'4	6218	188	3'0
October	7322	456	6'2	6191	194	3'1
November...	7222	488	6'7	6228	231	3'7
December...	7699	491	6'3	6672	258	3'8

From this it follows that in the first clinique the death-rate of the summer months was to that of the winter months as 3'0 to 6'7, and in the second clinique as 2 to 4, or about half as great.

At Bergen, according to Vogt,² there were 137 deaths from child-bed fever in the lying-in hospital and throughout the town during a period of thirteen years, the incidence in the several months being as follows:

Oct.	Nov.	Dec.	Jan.	Feb.	March	
15	21	21	20	15	11	Total, 94.
April	May	June	July	Aug.	Sept.	
10	5	7	9	8	2	Total, 41.

¹ 'Zeitschr. der Wiener Aerzte,' l. c.

² 'Norsk Magaz. for Laegevidensk,' 1872, iii, R. ii, 419.

If we take April to be in the cold season, the death-rate of the seven cold months is to that of the five warm months as 14·85 to 6·2.

I take the following figures from Lusk's¹ paper on the mortality from childbed fever in New York from 1867 to 1875.

Deaths from Puerperal Fever in New York during nine years.

December	.	.	.	173	} Winter	.	.	614
January	.	.	.	197				
February	.	.	.	244				
March	.	.	.	255	} Spring	.	.	694
April	.	.	.	236				
May	.	.	.	203				
June	.	.	.	136	} Summer	.	.	371
July	.	.	.	111				
August	.	.	.	124				
September	.	.	.	78	} Autumn	.	.	268
October	.	.	.	72				
November	.	.	.	118				
Total				1947

In the six cold months the deaths were 1223, and in the six warm months 724, the proportion being nearly the same as at Vienna and Bergen.

In the Paris Maternité, according to Hervieux,² there were, from 1830 to 1841, confinements to the number of 18,108 in the six cold months, and a mortality of 868 or 4·8 per cent.; in the six warm months the confinements numbered 15,956, and the mortality was 465 or 2·9 per cent. So that here again the proportion was the same as in the other cases.

§ 164. THE INFLUENCE OF COLD WEATHER AN INDIRECT ONE.

The prevalence of the disease as an epidemic or endemic during the cold months is often traced to the *kind of weather* proper to the season; or, in other words, the effects of cold weather, especially of cold and wet and of sudden changes of temperature, on lying-in women or women recently confined have been regarded as a material factor in the production of the disease. I consider this interpretation of the facts to be erroneous.

Not to mention that the malady has been as prevalent in mild weather as in extreme cold, in dry weather as in wet,

¹ L. c.

² L. c., p. 58.

in steady weather as in changeable, that doctrine is more especially opposed by two facts: firstly, the severest epidemics of puerperal fever have often been prevalent in one lying-in hospital of a city (Vienna, Berlin, London, Paris), while the other maternity hospitals of the place, experiencing the same kind of weather, have felt the disease to a very slight extent or not at all (a circumstance to which I shall recur); and, secondly, the women confined at the lying-in hospital of a town may be decimated by child-bed fever, while those confined at their own homes, and therefore not less exposed to the alleged harmful influence but probably more so (as among the poor) will have escaped the disease altogether. Nay more, the outbreak and prevalence of puerperal fever have been found to be much less dependent on the season of the year in private houses than in lying-in hospitals. Thus, Bradley remarks of the state of the weather during the prevalence of the disease at many places in Yorkshire in 1808-12: "It prevailed equally in cold and hot weather, in wet and dry seasons, in winter and summer." At Rostock in 1805 it occurred during the hot weather of summer, and there are several other epidemics in which a similar state of things may be discovered.

Many observers have arrived at the same opinion as to the influence of the weather on the production of the disease. Thus Cruveilhier, in his account of the epidemic at the Paris Maternité in 1830-31, says: "J'ai vainement cherché dans les vicissitudes atmosphériques, soit brusques, soit graduelles, dans le froid sec ou dans le froid humide, les causes de l'épidémie;" and Voillemier is led to the same conclusion by his experience of the 1838 epidemic in the Paris Clinique. In his report on puerperal fever at the Obstetric Institute of St. Petersburg, Grünwaldt says: "The weather had no influence at all, either in starting the sickness or in continuing it. A careful comparison of the state of the barometer and thermometer, and of the direction of the wind from day to day, showed that the cases occurred equally in all kinds of wind and weather, and showed also that the atmospheric conditions which obtained on the day of the child's birth had no constant relation to the subsequent course of the confinement."

Therefore, although we cannot doubt that epidemics of child-bed fever stand in some definite relation to the season, the cold months bringing a greater mortality than the warm; yet these differences cannot be explained by the direct effects

of the weather on lying-in or lately confined women ; and it is reasonable to suppose that it is the change in the hygienic condition of the lying-in hospitals brought about by the cold season which furnishes the real grounds for the rise of the sick-rate and death-rate. The influence of the season would, accordingly, be not a direct one, but an indirect ; and such is the conclusion to which Späth, Hervieux and others have come.

§ 165. KIND OF SOIL OF NO ACCOUNT.

It would hardly do for even the most enthusiastic advocates of the doctrine of the soil and the sub-soil water to make the *nature of the ground* answerable for the epidemic or endemic occurrence of puerperal fever. The disease has been prevalent at all elevations,—in lofty and dry localities as well as in low and damp,—on sandy or swampy soil as well as upon rocky ; and if there were any further doubt about the matter, it would be at once dispelled by the fact that, in the same institution, one division may be infested by the severest epidemics of child-bed fever for months together, while the women confined in another division, separated from the first only by the walls, are enjoying the best of health.

§ 166. SPECIALLY A DISEASE OF LYING-IN HOSPITALS.

Nothing in the history of puerperal fever, so far as we can follow it during the last three centuries, comes out so prominently and uniformly among the various factors with which the origin of the disease appears to be somehow bound up, than its *great prevalence in lying-in hospitals*, contrasting with its comparatively rare occurrence outside those institutions. Although it must be conceded, as we have already seen, that the cases of sickness and death from puerperal fever among women confined at their own homes, as given in statistics, are below the real number ; yet the difference between that number and the figures of the sick-rate and death-rate in lying-in hospitals is so great that the severest sceptic will not be

able to argue away the preponderance of child-bed fever in the latter. According to the statistical table given at p. 419, the deaths from puerperal fever in various civilised European countries, among women confined at their homes, averaged from 0·6 to 0·7 per cent. of the confinements; but in lying-in hospitals the average rises to 2 or 4 per cent., and under unfavorable circumstances it may rise even higher.

Lefort¹ calculates that of 934,781 women who were confined at their homes in various towns of Europe, 4405 or 0·47 per cent. died in child-bed; whereas among 888,312 women confined at lying-in hospitals, the deaths were 30,549 or 3·4 per cent. The mortality among puerperæ at St. Petersburg over a period of fifteen years was 0·7 per cent.; but in the Obstetric Institute of that City during the same period it was 3·8 per cent., and in the other lying-in hospitals it was as high as 4·6 per cent. These data are taken from the essay of Hugenberger,² who adds that, although the disease was six times epidemic in the Obstetric Institute during that period, not a single epidemic was observed in the liberties of the city. In the six great lying-in institutes of Paris (Maternité, Clinique, Hôtel Dieu, St. Louis, St. Antoine and Lariboisière) the mortality among puerperæ, according to observations extending over sixty years³ (1802-1862) averaged 4·8 per cent, whereas among women confined at their homes it was reckoned at not quite 0·6 per cent. In Genoa from 1857 to 1866 there died in child-bed only 0·42 per cent. of the women confined at their own homes, whereas the deaths at the lying-in hospital from 1855 to 1866 reached the enormous average of 8·8 per cent. At the maternity hospital of Pavia the deaths in child-bed averaged 2·3 per cent from 1861 to 1869. The lying-in hospital of Rome had the disease so regularly that it had to be closed almost every year on account of epidemic puerperal fever.⁴ Lastly it is a noteworthy fact that, of the 288 epidemics in the above table, 178 were exclusively in lying-in hospitals or other institutes for

¹ 'Gaz. des Hôpit.,' 1866, 152.

² 'Das Puerperalfieber,' &c., 48.

³ Tarnier, 'Gaz. des Hôpit.,' 1866, 151.

⁴ Sormani, l. c.

women in labour, 46 were in such institutions and more or less also among the women confined at their homes, 52 were in the towns or villages or their immediate vicinity, and 12 were widely prevalent over considerable tracts of country.

§ 167. RELATION TO OVER-CROWDING IN LYING-IN HOSPITALS.

It follows from this that puerperal fever has markedly the character of *a disease of hospitals*. Accordingly, when gynæcologists inquire into the cause of the disease, they always turn their attention to finding out the circumstances in lying-in hospitals with which the endemic persistence of the malady or its epidemic outbreaks appear to be associated.

Several of the earlier observers who occupied themselves particularly with this disease, such as Mercado, Willis and Sydenham, had recognised the fact that in child-bed fever they had to do with an infective process. According to the notions of the time, it was designated a "putrid" disease and was included in the class of "*febres putridæ*;" and that doctrine, of English and German origin, obtained a footing also in France after the overthrow of the system of Broussais. Meanwhile, the decomposition or putrefaction of animal or other organic matters had come to be recognised as an influence in the producing of "putrid" diseases (afterwards called "typhous"); and thus the conviction steadily gained ground that the source of the malady had to be looked for in the overcrowding of wards for lying-in or newly-confined women, and in the want of cleanliness and ventilation associated therewith, all the more so that the puerperal process itself gave rise to putrefying products of decomposition in abundance. The cause of the disease was supposed, accordingly, to arise from that mephitic state of things, in the form of a *miasma* which was suspended in the air and inhaled by the puerperal women; the toxic process thereby induced in them located itself chiefly in the genitals and the organs adjoining, as being the *pars minoris resistentiæ*. Experience showed that the introduction of one case of child-bed fever into a lying-in hospital or ward was quickly

followed by the appearance of the disease in other women who had been confined ; and from this it was inferred that the disease had the character of communicability, or that a *contagium* developed within the body of the patient, which was eliminated therefrom and caused the disease to spread independently of miasmatic influences.

Underlying this theory is the assumption that defects of hygiene, especially those arising from overcrowding of the lying-in wards, afford a real opportunity for the creation of epidemic or endemic foci of child-bed fever ; and that assumption has been received with hardly any objection in more recent times on the part of the medical directors of lying-in institutions. In the very earliest epidemiological notice of puerperal fever that we possess, the one relating to the Hôtel Dieu of Paris in 1664, it is pointed out that the hospital was crowded with patients at the time, an unusually large number of them being cases with wounds ; that the ventilation of the wards was extremely defective ; and, as expressly stated, that the lying-in department was directly over the wards occupied by the surgical cases. Cruveilhier writes to the same effect in his report on the epidemic of child-bed fever at the Paris Maternité in 1830 : “j’ai constamment vu,” he says, “les maladies prendre un caractère de gravité indomptable avec l’encombrement, ou s’atténuer par l’effet de la diminution de population.” In like manner Dubois and Voillemier speak in their accounts of the epidemic of 1838 at the Clinique, the former dwelling upon the unfortunate proximity of the institution to the dissecting rooms, and the latter remarking : “j’ai dit qu’outre les causes d’infection particulières à chaque maison d’accouchements, il en existait une autre, qui leur était commune à toutes, c’est la réunion d’un grand nombre de femmes accouchées sur un même point.”

Accounts of the same kind come from the Maternité of Lyons. Referring to the winter epidemics there in 1844-45 and 1845-46, Vernay says that the hospital was greatly overcrowded and that the ventilation was bad on account of the cold ; and as regards the epidemic in the summer of 1866, Fonteret tells us that, according to the very exhaustive inquiries of Dr. Delore, the medical director, the cause of

the epidemic had to be assigned to nothing but these internal sanitary defects. In the report by Cäderschjöld on an epidemic of child-bed fever in the Stockholm Lying-in Hospital in 1825, we find that the disease gained in extent just in proportion as the wards became crowded with patients; and that in subsequent years puerperal fever had broken out always when the hospital was particularly full. There is a very interesting paper by Retzius on the outbreak and progress of the disease in the same place in 1860 :

"Beds in the lying-in hospital," he says, "had been bespoken by an unusual number of women at the beginning of the year, and the number of patients that entered was more than the regulation allowed or than provision was made for. The admissions increased from day to day, to such an extent that neither the wards nor the bedding could be aired as they should have been." The consequences of this insanitary state of matters were soon shown in an epidemic outbreak of puerperal fever, of which we shall have to speak more particularly in the sequel; the disease, we are told, occurred "up to the end of March in the lower storey only, in the rooms set apart for the instruction of midwives. No lying-in woman who had a room to herself, with upwards of two thousand cubic feet of air, was attacked. In the common wards, which were adapted properly for three persons, it became necessary, in consequence of the pressure, to place four, so that the cubic space was reduced. Such an encroachment, although it may do no harm for a short period, cannot be endured for long, even supposing that perfect ventilation has been kept up all the time." The disease did not abate until vigorous measures to overcome these defects of hygiene were drawn up and carried into effect.

A state of things like the above has often been observed in the maternity hospitals of Dublin. Thus Clarke observes that the disease broke out in that city in 1787, at a time when the lying-in hospital was so crowded that they were sometimes obliged, "contrary to custom," to *put two women in one bed*. Douglas says that, according to his experience of the epidemics of 1810 and 1812-1813, we may assume with all certainty that "a very full maternity hospital and a quick succession of confinements has a very important influence on the production of child-bed fever." In like manner it broke out, in the new maternity hospital in Dublin, according to Beatty first in October, 1834, at a time when it was overcrowded. In Priestley's *résumé* of the recommendations on puerperal fever drawn up by the

Obstetrical Society of London, there is the following passage¹ relating to the present question : “ The influence of vitiated atmosphere in overcrowded hospitals in producing an endemic form of the disease is only too well confirmed. Whenever a number of lying-in women are aggregated together, there is danger that a miasm may be generated, which will develope puerperal fever, and it is by no means easy to define the amount of ventilation and isolation which are necessary to prevent these untoward consequences.”

Litzmann,² in his account of the epidemic of child-bed fever from 1840 to 1841 at the lying-in hospital of Halle, dwells upon the fact that the institution, all the time that the outbreak lasted, was unusually crowded with women about to be confined or newly confined, so that the airing and cleaning of the rooms could not be carried out to the extent that was to be wished. The same unfortunate condition of things was remarked in the epidemic of 1849 at the Stuttgart lying-in hospital ; also, in the maternity hospital of Bern in 1858, overcrowding preceded the epidemic, the disease being strictly limited at first to the two wards in which most of the newly confined women were (and in which they all spent the first few days after the child was born), while it did not show itself in the third ward until it became necessary to crowd the puerperæ into it also. If there were any further doubt about the importance of this etiological factor in the production of child-bed fever, it would disappear when we reflect on what is admitted by all, that nothing is more certain to bring an epidemic of puerperal fever to an end in a lying-in hospital than temporarily vacating the rooms where the disease has been, and thoroughly airing and cleaning them. Many of the older observers, such as Young, Clark, and Gooch, had often found this confirmed in their experience ; and there are still more numerous observations to the same effect from later times, such as those for Dublin in 1836 and 1837, for Vienna in 1792, for Copenhagen in 1839, for Kiel from 1834 to 1836, for St. Petersburg in 1858, and for Stockholm in 1825, 1840 and 1858. Writing of the Bellvue Hospital of New York, Reese³ says : “ A sudden

¹ ‘ Brit. Med. Journ.,’ 1876, Jan., p. 36.

² *l. c.*, p. 306.

³ ‘ Amer. Journ. of Med. Sc.,’ 1850, Jan., 99.

eruption of puerperal fever has appeared in the lying-in-wards several different times, the epidemic character of which has been in every instance promptly arrested by abruptly changing the apartments to another floor of the house, having a different exposure to the external air, and in which a due ventilation could be secured. The wards in which the fever appeared where meanwhile thoroughly cleansed and purified by white-washing &c.; and not until thoroughly aired and renewed by a change of furniture and bedding have they been again occupied."

Whenever puerperal fever has appeared in the Bellvue Hospital within the last ten or twenty years, according to Lusk's¹ account, the adoption of these measures has been attended with the same successful result. Harris² speaks in the same sense, from his experience in the lying-in department of the Pennsylvania Hospital of Philadelphia; and Hugenberger³ says:

"How much more the atmosphere of the hospital has to do with the production of an outbreak of puerperal fever than any other epidemical influence, was clearly proved in a negative way after the restriction of admissions and the closing of the lying-in hospital [of St. Petersburg] in 1846, 1848, and 1859; and the fact will be made clear to us, not only by the better condition of things always brought about after the wards, bedding, and furniture had been cleaned and disinfected, but also because the autumn months of those years in which the needed repairs and painting of the lying-in rooms, wards, and corridors had been done during the summer, were always the healthiest."

Furthermore, the factor of disease which we are now considering has been found to obtain, according to many observations, not only in lying-in hospitals but also in private houses. In the account of the groups of puerperal fever cases at Paris in 1746, we read that "*la maladie n'a attaqué que les pauvres femmes.*" Douglas says that, although no station in life is exempt from child-bed fever, the disease is decidedly more frequent among the poorer classes. In the Edinburgh epidemic of 1821, there were, it is true, some cases among puerperæ in comfortable circumstances; but the quarter of the town principally affected was

¹ 'Amer. Journ. of Obstetrics,' 1875, Nov., viii.

² 'Amer. Journ. of Med. Sc.,' 1847, Jan., 87.

³ L. c., 51.

where the poor lived. Twining and Webb are agreed in saying that the real reason why child-bed fever in India is more common among the Hindu women than among others, is the filth and the utter want of ventilation of the rooms in which they are confined.

“The woman after delivery,” says Webb, “is placed in a small damp room, very ill ventilated, with one small door only, no window or opening in the nature of a chimney. The door is always closed; the room is in a corner of the compound . . . and in a temporary hut of mats and bamboo, thatched with straw or grass . . . detached from the house, and generally kept for the purpose of the women of the family being delivered in it.”

This is perhaps the factor in the etiology that should help us most to understand what has been already mentioned, that lying-in hospitals have their visitations of puerperal fever oftenest in the cold seasons of the year. In the first place the number of persons resorting to these institutions is usually much greater in the cold months than in the warmer seasons, for reasons that are obvious; and therefore they are more frequently overcrowded at that time. In the second place, there are much greater difficulties in the way of sufficient ventilation during the winter months than at other times.

“There can hardly be a doubt any longer,” says Späth, “of the correctness of the principle above stated, that the winter months with their lower temperature have no direct ill effect on the state of health in lying-in hospitals. An injurious influence they do indeed exert, but I am convinced that it is by causing putrefying animal matters to collect in the building. For, on the one hand, it is impossible to keep the air in the rooms as pure as could be wished, owing to the want of ventilating appliances suited for an inclement season or bad weather. . . . On the other hand, there is usually a greater demand for accommodation in winter; and that is an additional and positive reason why decomposition-products accumulate.” Many other observers have expressed the same opinion.¹

If we compare the effect of these insanitary conditions upon the production of child-bed fever, with the significance of the same for the origin of erysipelas, we shall discover the most perfect resemblance between the two diseases as

¹ See the report on the epidemic of 1846 in the lying-in hospitals of Paris, and the papers by Vernay, of Lyons, and Lusk, of New York.

regards pathogenesis; and that resemblance comes out besides in the fact that child-bed fever, like erysipelas, is neither dependent of necessity upon the presence of these noxious things for its existence, nor is the very worst state of sanitation always followed by an outbreak of the disease. Over and over again has puerperal fever occurred in lying-in hospitals and in private life, both sporadically and in epidemics, without anyone being able to discover even a remote occasion for it in overcrowding, want of cleanliness, deficient ventilation or other noxious influence of the same class.

The following epidemics (or aggregates of cases) are examples of this: Heidelberg in 1811, the Paris Maternité in 1829, the Hôtel Dieu and other lying-in hospitals of Paris in 1831 and 1844, the Pennsylvania Hospital of Philadelphia in 1833, the lying-in hospital of Hanover in 1835, Schwezingen in 1863-65 and, in 1857, 1860 and 1861, and the newly-built and elegantly appointed lying-in institution at Munich, "in which every possible source of infection was not only avoided with the greatest care and watchfulness, nay even with painful anxiety in the building and internal arrangement of the hospital as well as in the service of the attendants, but was day and night looked for, guarded against and fought against in the case of each individual admission."¹

On the other hand, there are many lying-in hospitals which have escaped puerperal fever for years together, despite their having been often overcrowded. In the old Dublin Rotunda the disease broke out for the first time in 1767, or more than ten years after it was opened, and in the British Lying-in Hospital (London) not until 1760, or when it had been open eleven years. In his account of the epidemic of puerperal fever at the lying-in hospital of Bern in 1858, Hermann mentions that the institution had often been overcrowded before, without the sickness showing itself. Semmelweis gives statistics to prove that the extent of the disease in the Vienna lying-in hospital was by no means in direct proportion to the number of puerperal women taken in and tended. And there have been many other experiences of a like kind published by other observers. In estimating the importance of this etiological factor for the production of

¹ Martin (iii), l. c.

child-bed fever, just as for the genesis of erysipelas, we shall have to see in those insanitary conditions only a peculiarly favorable soil for the proper cause of the disease to develop in, or for the disease to spread in.

§ 168. EVIDENCE AGAINST THE THEORY OF A Miasmatic Origin.

It is this fact of puerperal fever seeming to break out not unfrequently quite apart from such external influences, that has given strong support to the theory of its *miasmatic or contagious-miasmatic origin*. It has been assumed that there is in child-bed fever a specific morbid poison, whether developed out of the puerperal process or otherwise produced, which is more or less widely diffused like the virus of scarlatina or typhus; this virus is taken into the blood in one way or another and brings about a general infection, which runs its course under some circumstances without forming local centres of disease (although usually the consequences are local lesions chiefly in the genital organs and the parts adjoining), and which gets transmitted (by contagion) from one person to another. The support for this theory was found, as we have seen, in the fact that in many cases no atmospheric, hygienic, or other noxious influences capable of accounting for an outbreak of the disease, could be detected. Another piece of evidence was that there had coincided with puerperal epidemics in the lying-in hospitals more or less numerous cases of the fever among women confined at their own homes; nay more, that it had grown into an epidemic in villages or towns quite independently of any occurrence of it in a lying-in hospital, that the epidemic had spread over considerable tracts of country, and had in some instances assumed even a pandemic character, breaking out at one and the same time in not a few parts of Europe. It is beyond the limits of my task to state here the endless variations upon this theme which have commended themselves to individual observers. I shall restrict myself to a presentation of facts, exclusively historical and epidemiological, which make against this doctrine of the origin of the disease

—a doctrine that has been accepted by prominent gynæcologists of the most recent period.

Firstly, in regard to the coincidence of the disease within lying-in hospitals and among puerperæ confined at their own homes, the facts in the above table show that such a coincidence has been observed only forty-six times in all out of two hundred and twenty-four epidemics; of these forty-six, it is expressly stated that in twenty-six the number of cases of puerperal fever outside the hospital was trifling; for five more there is no definite information as to the number of outside cases, doubtless because they were only here and there; and there are only fifteen epidemics of which it can be said that many cases were observed in private houses side by side with the epidemic diffusion of the disease in the lying-in hospital. I shall have occasion to show in the sequel that coincidences of the disease within and without the lying-in hospital, which would appear to have been much rarer in the last ten or twenty years than before, may be explained, for those cases where the coincidence was not a mere accident, in a perfectly satisfactory manner without being obliged to resort to the assumption of a morbid poison acting over a wide range, or of a “*constitutio epidemica*.”

How little justification there is for making that assumption under the particular circumstances, is proved by the history of epidemics which have been confined exclusively to lying-in hospitals. Numerous authorities dwell with special emphasis on the fact that many times during hospital epidemics of child-bed fever, often of a disastrous type and lasting for months, there had not been a single case among the women confined elsewhere than at the hospital—that the latter had in fact enjoyed singularly good health. Further, it has been often remarked that, in cities where malignant puerperal fever is epidemic every few years in one or more of the lying-in hospitals, the disease occurs comparatively seldom outside these institutions, and is never, or hardly ever, truly epidemic. This holds good for Dublin,¹ according to the infor-

¹ Of 19 epidemics recorded from 1760 to 1862, there were only 4 (in 1812-13, 1834, 1854, and 1861) in which it was observed that cases occurred among women confined at their homes, besides the cases in the hospital epidemic

mation of Clarke and Douglas, for Birmingham according to Ingleby, and it could be proved also for Vienna. As regards Coblenz we are told by Wegeler¹ that the oldest practitioners there could not call to mind a single epidemic of child-bed fever in the town, although every few years the disease attained a great height in the lying-in hospital. Hugenberger remarks that, during the period from 1845 to 1849, when puerperal fever was six times epidemic in the Obstetric Institute at St. Petersburg, the city itself had remained quite free from epidemics of it. But the most significant evidence against the "miasmatic" theory of the origin of puerperal fever is afforded by the fact brought out in the following tables, that in cities with more than one lying-in hospital, there has very rarely been any coincidence noticed between the epidemics in each; but on the other hand it has happened over and over again that one or more of these institutions have enjoyed good health while the disease has been making havoc in another; and that there have been the same differences between the various sections of a lying-in hospital separated from one another only by thin walls.²

Table of the Mortality from Child-bed Fever from year to year in various Lying-in Hospitals of the same city.

PARIS.

	Hôtel Dieu.	Pitié.	Margue- rite.	Hôpit. des Cliniq.	Maison d' Accouch.	Beaujon.	St. Louis.
1844	5'55 ⁰ / ₁₀	12'56 ⁰ / ₁₀	8'33 ⁰ / ₁₀	3'57 ⁰ / ₁₀	4'35 ⁰ / ₁₀	5'88 ⁰ / ₁₀	6'66 ⁰ / ₁₀
1845	5'01 "	8'33 "	3'22 "	3'33 "	3'70 "	6'25 "	3'84 "
1846	6'66 "	8'33 "	4'80 "	3'70 "	3'84 "	7'14 "	2'63 "
1847	3'57 "	11'11 "	—	2'38 "	3'22 "	1'72 "	1'78 "
1848	2'94 "	8'33 "	3'03 "	2'00 "	2'70 "	3'84 "	1'07 "

¹ 'Versuche einer med. Topogr. von Koblenz,' Kobl., 1835, p. 41.

² See Arnet's 'Geburtshülfe und Gynäkologie' (Wien, 1853, p. 47) with reference to the great lying-in hospital of Vienna.

ST. PETERSBURG.

	Obstetric Institute.	Training House.		Obstetric Institute.	Training House.
1845	2'3 ⁰ / ₁₀	6'4 ⁰ / ₁₀	1853	2'4 ⁰ / ₁₀	3'6 ⁰ / ₁₀
1846	4'7 „	4'0 „	1854	2'4 „	2'8 „
1847	2'2 „	2'2 „	1855	2'6 „	4'2 „
1848	6'3 „	4'6 „	1856	3'5 „	6'0 „
1849	3'4 „	5'8 „	1857	1'4 „	5'2 „
1850	2'8 „	8'4 „	1858	2'4 „	5'1 „
1851	1'2 „	5'3 „	1859	4'0 „	5'3 „
1852	2'5 „	8'9 „			

VIENNA.

	Lying-in Hosp. Division I.	Lying-in Hosp. Division II.		Lying-in Hosp. Division I.	Lying-in Hosp. Division II.
1833	5'29 ⁰ / ₁₀	2'26 ⁰ / ₁₀	1848	1'27 ⁰ / ₁₀	1'33 ⁰ / ₁₀
1834	7'71 „	8'60 „	1849	2'66 „	2'58 „
1835	5'55 „	4'99 „	1850	1'97 „	1'65 „
1836	7'47 „	7'84 „	1851	1'78 „	3'56 „
1837	9'09 „	6'99 „	1852	4'04 „	5'71 „
1838	3'04 „	4'94 „	1853	2'13 „	1'92 „
1839	5'42 „	4'52 „	1854	9'10 „	6'18 „
1840	9'24 „	2'65 „	1855	5'41 „	5'92 „
1841	7'80 „	3'52 „	1856	3'97 „	4'07 „
1842	15'75 „	7'59 „	1857	2'96 „	2'18 „
1843	8'95 „	5'98 „	1858	2'04 „	1'43 „
1844	8'23 „	2'30 „	1859	1'78 „	0'61 „
1845	6'90 „	2'03 „	1860	1'96 „	1'60 „
1846	11'44 „	2'79 „	1861	3'60 „	4'07 „
1847	5'04 „	0'96 „			

The instances of a succession of cases of child-bed fever among the residents of a locality at large, which have been designated by the somewhat exaggerated name of “epidemics,” have received important elucidations from the experience of the last half century or more. These experiences serve to prove that the dispersion of the disease depends on anything but a “puerperal miasma;” and, as I shall show in the sequel, they have furnished us with most valuable knowledge for the explanation of its real mode of origin and its diffusion. In the meantime we may remark of these

“epidemics,” that when they have occurred in large towns, as at Aberdeen in 1789 and Leeds in 1807, they have usually been confined within a small range or to one quarter of the town, not a single case of the sickness occurring among the lying-in women in adjoining districts.

Finally, as regards the simultaneous occurrence of child-bed fever in various parts of Europe (to which the experiences of the years 1781, 1819, 1825-26 and 1834-35 bear witness), there has been a theory of a quasi-pandemic diffusion of the disease deduced therefrom; but that deduction is based upon an arbitrary and erroneous association of heterogeneous elements. If the cases of puerperal fever occurring in groups had been more frequently reported than as a matter of fact they are, we should probably find hardly one year in which the disease had not been simultaneously prevalent in various parts of our continent to a notable extent; and the instances that now seem to be rare would lose much of their exceptional character. But even as the case stands, we are entitled to ask what right anyone has to conclude for the general operation of a common cause from the co-existence of a disease, say at Paris and Vienna, or at Dublin and Gratz. We have only to glance over the chronological table of puerperal fever epidemics, such as it is, to discover that there has been every few years a coincidence of that sort in the time of prevalence of the malady among a more or less considerable number of lying-in institutions. But we cannot seriously suppose that a disease which may have broken out in five or ten of the maternity hospitals of Europe, while there has not been a trace of a puerperal fever epidemic in other places or even in other hospitals of the same town, had owed its origin to a morbid cause generally diffused over wide tracts of country.

§ 169. EVIDENCE OF THE TRANSMISSION OF INFECTION.

An idea of much greater promise for the discovery of the origin of child-bed fever than all the tracing of it to a miasmatic source, is the one that was long ago indicated by

Willis,¹ was afterwards developed by writers such as Eisenmann and Helm, and in recent years has grown to be a well-established theory—the idea, namely, that *puerperal fever is a septic or infective traumatic malady*. The evidence for the correctness of that view, adduced by me in the presentation of facts in the first edition of this work, was accepted almost unanimously by German gynæcologists. I am justified, therefore, in making a somewhat detailed statement of the data on which the proof rests; and I shall select the weightiest of the observations which I collected originally, adding to them, by way of further confirmation, such of the more recent as are specially worthy of note.

(1) DENMAN ('Introduction to the Practice of Midwifery,' Lond., 1788, ii chap., 19) was the first, so far as I know, to allege that childbed fever was sometimes carried by doctors and midwives, who had been in attendance on puerperal fever patients, to other lying-in women.

(2) GORDON ('Treatise on the Epidemic Puerperal Fever of Aberdeen,' Lond., 1795), in his account of the Aberdeen epidemic of 1789-92, gives several cases where midwives or nurses in attendance on patients with puerperal fever, had carried away loeal secretion on their hands, and had given the disease to the lying-in women whom they next had to deliver. In the same way the infection was carried from Aberdeen to a woman who lived at Fintray; and the midwife who delivered her, and attended her through the fever, gave the infection to two other women in childbed in the same parish.

(3) ARMSTRONG (see List of Writers), in his account of the epidemic of puerperal fever in Northumberland in 1813-14, has the following: "It is a singular fact that, in whatever place the fever in question occurred, it was principally limited to the practice of one accoucheur in that place. To adduce an example in point, Mr. Gregson attended, with three solitary exceptions, all the women who were afflicted with the puerperal fever at Sunderland, and that gentleman has, with a liberality which does him the greatest credit, declared that in his practice the fever was excited and kept up by contagion."

(4) DOUGLAS (l. c.) says: "I know that during one of the epidemics [in the Rotunda] an accoucheur attended several women [in Dublin] in their confinement, who all took puerperal fever and died. . . . The young man was so afraid that he had introduced something contagious that he would attend no other crying woman while the epidemic lasted."

(5) GOOCH ('Account of some of the most important Diseases peculiar

¹ Enumerating the "causæ evidentes" of "febris puerperalium putrida," Willis says (l. c., 129): "Huc faciunt partus laboriosus, circa uterum unitas soluta, contusio, rerum praeternaturalium retentio, dispositio ulcerosa et pleraque alia accidentia, quae necessitate quadam inducuntur."

to Women,' Lond., 1829, p. 4) says: "There is still another remarkable circumstance in the prevalent or epidemic form of this disease. It is not uncommon for the greater number of cases to occur in the practice of one man, whilst the other practitioners of the neighbourhood, who are not more skilful or more busy, meet with few or none. A practitioner opened the body of a woman who had died of puerperal fever, and continued to wear the same clothes. A lady whom he delivered a few days afterwards was attacked with and died of a similar disease; two more of his lying-in patients, in rapid succession, met with the same fate. Struck by the thought that he might have carried the contagion in his clothes, he instantly changed them, and met with no more cases of the kind. A woman in the country, who was employed as a washerwoman and nurse, washed the linen of one who had died of puerperal fever; the next lying-in patient she nursed died of the same disease; a third nursed by her met with the same fate, till the neighbourhood, getting afraid of her, ceased to employ her."

(6) ROBERTON ('Med. Gazette,' ix, 1831-32, p. 503), in his account of the Manchester epidemic of 1830, says: "Mrs. A. B., a midwife in great practice among the patients of the [Manchester Lying-in] Charity, had on the 4th of the preceding month (Dec., 1830) delivered a poor woman, who soon died with symptoms of puerperal fever. From this date to the 4th of Jan. inclusive—exactly one month—this midwife delivered thirty women residing in different parts of an extensive suburb, of which number sixteen caught the disease, and all of them ultimately died. These were the only cases of puerperal fever which had for a considerable time occurred in Manchester. The midwives, commonly twenty-five in number, deliver on an average ninety women per week, which is about three hundred and eighty in a month. Now, of this number delivered during the month in question, none had puerperal fever except the patients of Mrs. A. B. Yet all this time this woman was crossing the other midwives in every direction, scores of the patients of the Charity being delivered by them in the very same quarters where her cases of fever were happening.

"The decision of the medical officers of the Charity was to the effect that Mrs. A. B. should abandon her practice for a short period and go to the country. In a short time after this meeting, cases of puerperal fever among the patients of other midwives, as well as in private practice, began to appear in various parts of the town. In the course of the spring months a great number of women died of this fever. It never prevailed more generally, nor perhaps even more fatally, in Manchester. By about the beginning of June it had disappeared. . . . That the fever was occasionally conveyed *directly* from the diseased to the whole, I possess other evidence than I have stated.

"In one instance, within my knowledge, a practitioner introduced the catheter in the case of a poor woman labouring under puerperal fever, late in the evening; and in the course of the said night he had to attend a lady in her confinement a little way in the country. On the morning of the second day after delivery, this lady had a violent rigor and

the other early symptoms of the malady. In another instance a surgeon was called, while in the act of inspecting the body of a woman who died of this fever, to attend a labour; within forty-eight hours after being put to bed, the woman was seized with the fever."

(7) CAMPBELL ('Med. Gaz.,' ix, 1831, p. 354), writing of the puerperal fever in Edinburgh, says: "In October, 1821, I assisted at the dissection of a woman who died of the disease, after an abortion of the early months; the pelvic viscera, with the external coats were removed, and I carried them in my pocket to the class-room. The same evening, without changing my clothes, I attended the delivery of a poor woman in the Canongate; she died. Next morning I went in the same clothes to assist some of my pupils who were engaged with a woman in Bridewell, whom I delivered with forceps; she died. And of many others who were seized with the disease within a few weeks, three others shared the same fate in succession."

(8) HUTCHINSON mentions the following fact: Two doctors residing ten miles apart met in consultation over a patient with phlegmonous erysipelas who lived at a place midway. At their visit each of them made a free examination of the affected limb and its discharging surface with his hands; within the next thirty or forty hours each of them delivered a woman in his own district, and both women took puerperal fever and died.

(9) INGLEBY (l. c.) is the authority for the following: In 1833 a friend of his in practice in Birmingham attended a lady for phlegmonous erysipelas, and found it necessary to make free incisions into the inflamed part. On the 28th August, at six in the evening, having just done that operation, he attended a lady in her confinement, who was seized with puerperal fever two days after, and died. Another patient, whom he confined the same evening, shared the same fate (Case II). On the 3rd September, or two or three days after the first fatal case, he attended a third labour, and in that case also the woman died of childbed fever on the third day of her confinement. On Sept. 4 another labour, and again puerperal fever, which in this case fortunately ended in recovery. On Sept. 5 the doctor, with his assistant, made an examination of the body in Case II, and each of them then went without changing his clothes to attend a case of labour; both the women took puerperal fever, and one of them died. Still wearing the same clothes, the doctor delivered yet another woman on the 7th September, and she too died of puerperal fever five days after. Several slighter cases of the disease followed, and he then laid aside his midwifery practice for a time, wherewith the "epidemic" came to an end.

Another practitioner, who had also been making free incisions into a patient's arm for phlegmonous erysipelas, was called half an hour after to a woman in labour. He found placenta prævia and did the operation necessary; next day the woman took puerperal fever and died. Six or seven hours after that confinement, the same practitioner was called to another case of labour; here again childbed fever appeared the day after, but this time it ran a favorable course. In November, 1836,

Dr. Ingleby was present at the necropsy of a woman dead of puerperal fever, when the practitioner whose case it was told him that he had opened several abscesses shortly before he went to deliver the woman, and that she became ill on the day after her confinement. Dr. Ingleby cautioned him of the risks he incurred, and enjoined him to keep an eye on the other patients whom he had delivered during the last few days. This was on Thursday morning, and on Saturday he came to Dr. Ingleby to say that three more of the women whom he had attended in labour had taken puerperal fever, two of them having been confined on the Tuesday and the other on Thursday. Cases V and VI, which both ended fatally, occurred on the Monday following, and there was a seventh case, which fortunately recovered. This practitioner then gave up his midwifery practice for a time, and the "epidemic" ceased.

(10) CLARK ('Med. Gazette,' v, 1847, p. 331) attended two women in labour in May, 1847, at eight days' interval; both of them took puerperal fever and died. When the first case occurred, he thought that the noxious influences were purely local, in the dwelling, &c., of the patient. But after the second case, he found out the real cause. On the day that he delivered the first woman, he had made free incisions into the arm of a sailor who had been admitted into the Colchester Union Workhouse with phlegmonous erysipelas; he had then gone to attend the labour, and had probably brought the infection to the patient. He at once gave up his midwifery practice for a time; and, besides those two cases, no more puerperal fever occurred at that time in Colchester.

(11) STORRS ('Prov. Med. Journ.,' 1842, March, No. 15) gives the following history of the Doncaster epidemic of 1841: "During the whole of the winter of 1840-41, erysipelas, typhus fever, and scarlatina of a malignant form, prevailed in Doncaster to an unusual extent, especially erysipelas, which I have never before known to be so general or severe. Puerperal fever was never known to have prevailed epidemically up to this time, or if it did so, it was never acknowledged. On the night of January 7th, or early on the morning of the 8th, the most severe night of the winter, when the thermometer was lower than it had been for many years, I attended Mrs. D. (Case I), a hard-working washerwoman, with her tenth child. Her labour was perfectly natural, though rather more severe than she was accustomed to, and she had some severe rigors previous to delivery, which I ascribed to the severity of the night. On the morning of the 9th, thirty hours after delivery, she was seized with another severe rigor, succeeded by severe abdominal pain, excessively rapid pulse, and all the symptoms ascribed to puerperal fever in its severe form. She died on the morning of the 12th. [Then follow seven other fatal cases from 17th Jan. to 27th Feb.] Being now led to suspect that some extra-*puerperal* causes produced the mischief, I mentioned the cases which I was most inclined to blame to Dr. Thompson, of Sheffield, who confirmed me in the belief that the fever had probably sprung from them. One was a case which had been in the commencement gangrenous erysipelas of the leg and foot in a

stout, gross woman. On looking back at this case I recollected that I had been called to her on the very evening prior to my attending Mrs. D. (Case I). . . . I left off the practice for a month, and am happy to say that since that time I have had but one case to cause me any uneasiness."

(12) STORRS ('Prov. Med. Journ.,' 1843, Dec., p. 163) gives also the following facts communicated to him by friends: Dr. Reedal, of Sheffield, undertook the treatment of a young man with a suppurating bubo, which had taken on a phagedænic character. He had never had a case of puerperal fever in his practice before, and no case of the sort had been seen in the town; but between the 27th October, the day on which he began to treat the bubo, and the 3rd November, five women delivered by him took puerperal fever and died; in a sixth case the confinement progressed favorably, and in a seventh the attack of puerperal fever was mild. Dr. Reedal pointed out that the fatalities had happened to women whom he had been called to after bandaging his bubo-patient, that they occurred in the most diverse parts of the town, and that as soon as he ceased attendance on the patient with the bubo, he had no more cases of puerperal fever in his practice to deplore.

Three practitioners who took part in the examination of a person dead of a strangulated hernia, which was found to be gangrenous, were shortly after in attendance on a number of women in labour, several of whom took puerperal fever and died. Thereupon they gave up their midwifery practice for a time, and had no more misfortunes of that kind among their lying-in patients to blame themselves for.

(13) LEE ('Med. Gazette,' 1843, Aug., p. 755) relates the following: A doctor in the vicinity of London made an examination on the 16th of March of the body of a woman dead of puerperal peritonitis; between that date and the 6th April, three women confined by him took puerperal fever. Lee himself went straight from a post-mortem on a case of puerperal fever to a woman in labour, who also took the disease. In December, 1830, a midwife of the British Lying-in Hospital, who had two cases of puerperal fever under her charge, made an examination of a pregnant woman, whose labour shortly began, and who sickened and died the day after she was delivered.

A practitioner in the west end of London, who had to treat a case of phlegmonous erysipelas of the leg with a great amount of discharge, lost three of his midwifery patients at that same time from puerperal fever. Lee assisted him at the examination of one of the bodies, and, in spite of every precaution, the two next cases that he attended in labour took the fever and died. The same misfortune happened to Lee several times afterwards.

(14) ELKINGTON ('Prov. Med. Journ.,' 1844, January, p. 287) narrates the following facts concerning the Birmingham epidemic of 1833: "My attention was first directed to puerperal fever as an epidemic in 1833. On the 28th of Aug., 1833, after visiting a bad case of erysipelas at the edge of the town, and making free incisions through the diseased part, I attended Mrs. J., living in the centre of the town, who, after a

favorable labour, was confined of her second child. She was doing well until the evening of the third day, Aug. 31, when she was attacked with fever and died Sept. 3. On Aug. 28 also I attended Mrs. C. of her first child, and went directly from attending Mrs. J. Mrs. C. had a severe labour, followed by flooding, and was feverish and poorly from the first day. She was taken worse on the 30th, and died Sept. 4. On Sept. 3 I attended Mrs. E. of her third child. She had a favorable labour, and went on well till the 5th. She was then attacked with fever and died on the 9th. On Sept. 5 I examined the body of Mrs. C., the second patient, assisted by my brother. As we were leaving the house, we received each a message to attend a labour. Mrs. W., my brother's patient, was attacked on the 8th, and died on the 11th. Mrs. Y., the person I attended, was also attacked on the 8th, but recovered."

(15) BLACKMORE (l. c.), in his account of the epidemic of puerperal fever at Plymouth in 1831, says: "Case I of my series was the second of eight or more cases of puerperal fever that occurred in the practice of one accoucheur within a fortnight; during the next fortnight he had seven more cases, which all ended fatally; in the week following at least three women delivered by him took the fever, of whom two died; and several cases occurred in his practice subsequently. I make out that in the practice of this medical man at least eighteen cases of puerperal fever occurred in rapid succession, most of them at a time when all the other practitioners of the town had not met with a single case of the disease. Scarcely had the malady ceased among his patients, when a second and a third accoucheur had cases of puerperal fever, which were only the beginning of a long series. Between those three series of cases there was no communication whatsoever; none of the three practitioners had visited the patients of any of his colleagues. The accoucheur was the only medium, so far as was known, of spreading the disease among his patients."

(16) SIMPSON ('Edin. Monthly Journ. of Med.,' 1851, July) relates the following: In the winter of 1836-37, Dr. Sidey had five or six fatal cases of puerperal fever in his practice in rapid succession, no other practitioner in Edinburgh having had any. Simpson attended the examination of the body in two of the cases, and took the diseased parts into his hands so as to examine them more closely. The next four women delivered by himself took puerperal fever, and these were the first cases of the disease that he had ever had in his private practice. Peddie (ibid.) tells us further that the communication of the disease was not confined to the cases mentioned by Simpson; a practitioner in Leith, who had examined a piece of the uterus which Simpson had brought home with him, had three cases of puerperal fever among his patients directly after. Another instance given by Simpson is the following: A doctor in Leith made a post-mortem examination of a woman with pelvic abscess; within the next fifty hours he was called to five cases of midwifery; in four of these puerperal fever came on; and in the single case which did well, the child had been born before he

arrived. Another instance was told to Simpson by Dr. Patterson: A doctor had several cases of puerperal fever in his practice one after the other, whereupon he changed his clothes as a precaution; the women whom he delivered after that did well, until he happened to put on a pair of gloves which he had been wearing at the time when he was attending the first-mentioned cases, and immediately the disease reappeared among his lying-in patients.

(17) FENTON ('Brit. Med. Journ.,' 1875, Feb., p. 208) gives an official report upon a small epidemic of puerperal fever at Coventry, which was due to the linen that had been used by lying-in women with puerperal fever at two charitable institutions having been given to two other women about to be confined; these women took puerperal fever, and the midwives who attended them carried the infection to other cases.

(18) PUNCH ('Allgem. Annalen der Heilkunst,' 1811, p. 329) observes that most of the cases of puerperal fever observed in 1810-11 in the small Saxon town of Landsberg happened in the practice of one midwife; after this woman ceased to attend midwifery cases, nothing more was seen of the disease.

(19) LITZMANN (l. c., p. 308) narrates the following in connexion with the epidemic of 1841-42 in the lying-in hospital of Halle: "The disease was not epidemic in the town itself nor in the vicinity; but in the month of February two women suddenly took it in the town and died, and one woman in the country. All three were delivered by myself, two with the forceps, and the other by *accouchement forcé* owing to placenta prævia. The symptoms and course of the malady corresponded exactly to what had been observed at the Institution, and I do not hesitate to assume a transmission of contagion. From that time all the lying-in women escaped the disease, although operative interference was required for several of them. Besides those three cases I could learn of no case of childbed fever at that time outside the Institution."

(20) KIRCHOFF, in his account of a small epidemic of puerperal fever at Leer in 1852, says that nearly all the cases occurred in his own practice or in that of the district surgeon, no serious childbed maladies having been reported by the third practitioner or by the midwives of the district.

(21) MARTIN ('Monatschrift für Geburtskunde,' x, 259) relates the following incident in the epidemic of 1856 at the lying-in hospital of Munich: "For a considerable time, in January and February, there had been a cessation of cases of serious illness among the patients in the lying-in hospital; when suddenly two women on the same day fell ill with symptoms of the epidemic puerperal fever. They had been delivered on the same day and almost at the same hour; but for neither of them could an obvious cause of the sickness be found, which would not apply to the rest of the inmates. At length, after prolonged inquiries into this remarkable occurrence, it came out that an assistant, without the knowledge of the director, had opened the body of a child in the deadhouse some distance off; and, having carefully washed (as he

said) with chlorinated water, he went and made an examination of those two women, but of no others. As both of these women became ill at an unusually early period after the labour, and as they were the only two attacked of all the lying-in women in the house, the assistant admitted that he was to blame. He added that he had done the same thing in December, on the day when puerperal fever first appeared in the institution; and on that occasion also it was only those women whom he had examined after making the post-mortem examination who fell ill in the first instance."

(22) SCHULTEN ('Virchow's Archiv,' 1859, xvii, 228) gives an account of epidemics of puerperal fever at two villages in Rhenish Hesse, where the disease was carried by a midwife from one lying-in woman to others; in both instances the epidemic ceased as soon as the suspected midwife gave up her duties for a time.

(23) WEGSCHEIDER ('Monatschr. für Geburtskde.,' 1864, Feb.) records the following observations: A midwife in Berlin named L. delivered a woman, who took puerperal fever and died. Three days after she attended another woman in labour, who also took the disease and died. After refraining from midwifery practice for a short time she undertook three confinements in quick succession, which were all followed by puerperal fever. She now gave up her work for several weeks, religiously discarded her linen, clothes, and instruments, and replaced them with new; and after that had no more of these cases in her practice. A second series of observations is even more remarkable. A midwife named R. delivered four women in one day, who all took puerperal fever and died; on that day the midwife herself had been suffering from commencing rose in the face, and she was afterwards so ill with it that she had to keep her bed for a fortnight, and was not able to resume her work for nearly a week longer. Dr. Wegscheider seriously cautioned the woman not to begin practice again until she had provided herself with new clothes and instruments. However, she gave no heed to this counsel, and resumed work on the 21st October, or nineteen days from the date of the first case that went wrong. Thereupon a woman delivered by her on Oct. 21st fell ill, then two women on the 25th, another on the 30th, another on the 31st, two on the 2nd Nov., and two on the 4th; so that of twenty-two puerperæ whom this midwife attended between the 21st Oct. and the 4th Nov., nine fell ill and seven died; and of the thirteen who did well nearly all were multiparæ, with whom the midwife remained only a short time, or found the child born before she came. In judging of that instance the following things should also be taken into consideration: the cases occurred over a very wide radius of Berlin, in the most diverse parts of the city; at the same time, so far as could be ascertained, only one other of the numerous midwives practising in the same quarters had cases of puerperal fever (three in rapid succession); so that there could not be the remotest idea of a general diffusion of it throughout the city.

(24) MAIR ('Bayer. ärztl. Intelligenzbl.,' 1865, No. 19, p. 269) gives the case of a midwife in Munich, who attended a woman with a severe

attack of puerperal peritonitis, and infected four puerperæ within three weeks.

(25) WERDMÜLLER ('*Monatschr. für Geburtskunde*,' 1865, p. 293) narrates the history of an epidemic of puerperal fever in the commune of Maur (Zürich), in which the first person to fall ill had probably been infected through a woman just returned to her home from the lying-in hospital, where she had suffered from childbed fever. The next case was the sister-in-law of the former, and she used the same delivery-stool and had the same nurse. The midwife went straight from this patient to a crying woman in the neighbourhood, who was likewise infected; and in this way the epidemic was prolonged for several months by infection from one lying-in woman to another.

(26) STEHBERGER ('*Monatschr. für Geburtskunde*,' 1866, April) relates that the puerperal fever in Mannheim, which had been very rare previously, and in sporadic cases only (one to three cases in the year), rose to such a height in the spring of 1865 that thirteen patients died of it in four months. All these cases happened in the practice of two midwives; whereas the other twelve midwives in the town and the whole of the medical practitioners had only one case of the disease at that time among them, being a case in which craniotomy had been performed, and from which the puerperal fever did not spread.

(27) KAUFMANN gives an account of a slight epidemic of puerperal fever at Dürkheim in 1866; five women in childbed were involved in it, who had been delivered by the same midwife one after the other within the space of a few weeks.

(28) HÄHNLE says concerning the Schweningen epidemic of 1879, that the whole of the fourteen cases (among thirty-four confinements) occurred in the practice of one midwife, while the other midwife in the place, who had just as much to do, had not a single case to report.

(29) HUGENBERGER (l. c., p. 49) writes: "Having made a post-mortem examination immediately before, Dr. Etlinger (in 1847) infected a woman in labour in the institution [St. Petersburg Obstetric Institute], and a lady in the city whom he had occasion to examine after abortion; both died of pyæmia. Twice, under the same circumstances, I had myself the misfortune to infect lying-in women with cadaveric poison in making an examination or in removing the after-birth. Although we have taken the greatest care about the examination of dead bodies at our institution since that time, it cannot be doubted that many other casualties beyond our control have occurred through the negligence of the service."

(30) CÄDERSCHJÖLD (l. c.) points out, in his report on the epidemic of 1825-26 at the Stockholm lying-in hospital, that Dr. Idström, having made an examination of the body of a patient dead of puerperal fever, saw that disease ensue in five successive cases of labour which he attended outside the hospital.

(31) CÄDERSCHJÖLD ('*Svenska Läkare Sällskapets nya Handl.*,' ii, 32) also narrates the following incident in connexion with the general lying-in hospital of Stockholm in 1832: "On the 27th October a

patient who had been delivered with the forceps took puerperal fever. The midwife who attended this case delivered two women between the 28th and 31st October, both of whom took puerperal fever and died; at the same time three women delivered by other midwives did well. This incident gave new vitality to the idea that puerperal fever may be propagated under certain circumstances by contagion; and in the inquiry that followed, it came out that there had been neglect of a rule, introduced several years before, to the effect that each patient should be washed with her own sponge assigned to the particular bed, and dried with her own towel, and that the sponge was to be boiled before it was used for another lying-in patient. This rule was now revived, and there were no more cases of the disease until the 17th of November." An incident of very much the same sort is related by Elliot (*ib.*, iii, 238) of the same lying-in hospital in 1839.

(32) SPEYER, writing of the epidemic of childbed fever at Aalborg in 1844, says that, of twenty-two women who were confined there between the 6th Nov. and 12th Dec., eight took the disease and seven died. Of the whole twenty-two cases, seventeen were attended by one midwife and five by another; all the cases of sickness and death occurred in the practice of the former, following one another at intervals of from three to fifteen days. Speyer lays special stress on the fact that this midwife paid the greatest attention to her puerperal-fever cases, remaining with them as long as her time permitted, and going straight from them to other women in labour when she was called to them.

(33) STAGE ('Undersøgelser angaaende Barselsebern i Danmark udenfor Kjøbenhavn,' Kjöb., 1868) gives an account of a number of small epidemics of puerperal fever in various towns and country districts of Denmark. In 1850 an epidemic of that kind sprang up in the vicinity of Aarhus; nine women were attacked, who had all been delivered by the same midwife; whereas the other midwife of the place did not have a single case. The same thing was observed in 1855 in the case of five women at Skibby, in 1859 on the island of Bogoe, at Frederickshavn in 1862, and Erdboebrug in 1864. In the last of these, the infection probably started from a case with pyæmic abscesses, which the midwife had tended with special care. On Langeland in 1864, all the women delivered by one midwife between the 29th June and the 18th July, nine in number, were seized with puerperal fever. Besides these, one other lying-in woman took the fever, having been attended by another midwife, who had administered an enema to one of the first series of cases, and had directly after used the same syringe for her own patient. On the first midwife ceasing from practice the epidemic stopped.

(34) GRISAR ('Bull de l'Acad. de méd. de Belgique,' 1864, No. 7) has the following: In the end of December, 1842, he attended a woman in labour, who took puerperal fever and died. From that date to the following March, out of sixty-four women delivered by him, sixteen had the fever and eleven died of it; meanwhile there were no cases of puerperal fever in the practice of his colleagues. The same thing

happened again in his practice towards the end of 1862; of nine women whom he confined between the 5th Dec., 1862, and 26th Jan., 1863, eight had the disease and four died of it.

(35) VOILLEMIER records the following: M. Depaul, interne in the Paris Maternité, was called to attend a lady in the town, at a time when there was an epidemic of childbed fever in the lying-in hospital. He had made an examination of one of the dead bodies shortly before going out to his patient, who took puerperal fever in due course and died.

(36) ARNETH ('Ueber Geburtshülfe und Gynäkologie,' Wien, 1853, p. 52) mentions a case told to him by Dubois, in which a medical friend of the latter, who carried on a small lying-in hospital in the country, infected two women with fatal puerperal fever, the reason of which was, as he seemed to think established beyond all doubt, that he had previously made a post-mortem examination. Since that time Dubois gets women from the town to come for the examination-practice by paying them, so as to ensure that these examinations shall not be made on women who are on the point of being confined.

(37) ROBOLOTTI ('Giornale della soc. italiana d'igiene,' 1879, No. 6, p. 700) gives a number of instances at various places in Lombardy, in which puerperal fever was epidemic among the patients of one medical practitioner or of one midwife; as at Cicognolo in 1870-73, Rivarolo in 1874, Scandolara in 1878, and Olmeneta in 1879.

(38) RIGLER (l. c., p. 240), referring to the cases of childbed fever which he had himself seen in Constantinople, says: "There has never been a contagion of the volatile kind observed; but we are inclined to think that contact of the pudenda of a woman in childbed by hands not absolutely cleansed from the discharges of a septic patient may have serious consequences."

(39) PALEY ('Med. Gaz.,' Dec., 1839, vol. i, n. s., p. 397) writes as follows from Halifax: "During my attendance [in consultation] on the first case [gangrene of the scrotum] one morning, whilst the surgeon was dressing the patient, the scrotum and penis being in a gangrenous state, a messenger came to request him to go to a woman in labour, who resided about half a mile from our patient; and he obeyed the summons without loss of time. Four or five days after this, on meeting again, he said, 'You will recollect that I was sent for to a woman in labour on such a day.' I replied, 'Yes; what of that?' 'She is dead; everything seemed to be going on well until yesterday, when she was seized with violent pain in the region of the uterus, and she died before I had time to do anything to relieve her.' In the course of two or three days, on meeting again, he said, 'It is very odd, Dr. Paley, I have lost another patient in the same unaccountable way as before;' and the next morning, at our meeting, he stated that he had another patient, about two miles off, seized in the same manner, whom he requested me to visit along with him. After seeing his patient, I told him that she was labouring under puerperal fever, and before we left the house he was sent for to visit another woman whom he had attended in labour in the same village. I accompanied him, and found her also the subject

of puerperal fever. I believe that he had in all six cases of this disease. . . . There is not the slightest doubt on my mind that the surgeon who was in attendance was the means of communicating something (call it what you please) from the patient labouring under the disease of the scrotum to the lying-in women, which in them produced puerperal fever. . . . I pointed out these circumstances to the surgeon, and, at the same time, advised him to go from home two or three weeks, and to have his clothes washed and fumigated. He did so, and the plague (for such it seemed) ceased."

(40) WARRINGTON ('Transact. of the Coll. of Phys. of Philad.,' 1842) stated in the course of a discussion on the conveyance of puerperal fever by third parties, that he had confined three women in quick succession after having made an examination of the body of a patient dead of puerperal peritonitis, and they all took puerperal fever one after the other. In like manner West said that, under the same circumstances, seven patients delivered by Dr. Jackson all took puerperal fever, and five of them died.

(41) HOLMES ('New Engl. Pract. Journ. of Med.,' 1843, April, p. 503) gives the following cases: A doctor examined the body of a man who had died of gangrene of the leg, and next day he attended a labour; this woman, and six others whom he delivered in the time immediately following, took puerperal fever. Another doctor, who had five cases of puerperal fever in quick succession, writes to Holmes that he had occasion to treat a patient with malignant erysipelas just before the first of these cases, and that he had probably been himself the medium of spreading the disease.

(42) LEASURE ('Amer. Journ. of Med. Sc.,' 1856, Jan., p. 45) says: that when malignant erysipelas was prevalent at Newcastle, Pennsylvania, in 1852, all the cases of midwifery in his own practice and in that of another doctor got puerperal fever; and he expresses his conviction that they had themselves carried infection to the women in labour. Many other observations of the same kind are recorded by Holston, Galbraith, Minor, Ridley, and other American practitioners for the period of the great erysipelas epidemic; and to these I shall return later on.

(43) PERKINS ('New York Journ. of Med.,' 1852, May, p. 330), in his account of the epidemic of 1850 at a New York lying-in hospital, says: "As to the cause of the endemic, I have every reason to believe that a post-mortem examination of the body of Mary Murray, who was brought into the hospital on the 25th November, and who died in an hour after admission of a peritonitis, which had resulted in a purulent effusion, was (through the necessities of the case) the prime cause of the endemic which followed. . . . The first case which occurred was delivered by the same hand which made the autopsy, on the afternoon of the day on the evening of which the woman was delivered."

(44) LEVERGOOD ('North Amer. Med.-Chirurg. Rev.,' 1857) relates that Dr. Lloyd, being then engaged in the surgical treatment of a case of phlegmonous erysipelas, delivered three women, who took puerperal

fever and died, although there was not the faintest trace of epidemical influence to be discovered, nor anything amiss in the hygienic surroundings; at the same time the patients delivered by other practitioners passed through childbed without mishap. Dr. Lloyd, who was in much request as an accoucheur, transferred his erysipelas case, and had no misfortunes in his midwifery practice after that.

(45) FÉRIS ('Arch. de méd. nav.,' 1879, Oct., p. 253), in his account of the disastrous prevalence of puerperal fever at Monte Video in 1875, states that the number of cases diminished considerably after the midwives in attendance on patients with childbed fever had been forbidden to wait on other women in labour or lately confined.

(46) HALL (l. c.), writing from Tasmania, says that if a practitioner has had to treat a case of puerperal fever, he may expect, just as in the Old World, soon to have other cases of the same disease in his practice.

All these observations, which I could easily have trebled, are taken from the most diverse parts of the world, and they all tell one way. The significance which they seem to me unquestionably to possess is in proving experimentally the origin of puerperal fever by direct conveyance of a noxious substance, without any need or even justification for assuming that there had been influences of a general kind at work in the pathogenesis. It is the great merit of Semmelweiss, as I have already mentioned, to have solved the problem in this sense by exact research; and his merit will not be lessened by the fact that, in forming his conclusions from the data furnished by the lying-in hospital of Vienna, he took up a one-sided point of view.

The statistics of confinements, and of deaths from childbed fever, in the great Maternity Hospital of Vienna during a space of sixty-two years (1800—1861), tend to show that the rate of mortality in that time may be differentiated according to three well-marked periods:

	Confinements.	Deaths from puerperal fever.	Death-rate per cent.
First period (1800—1822)	. 47,409	... 683	... 1.44
Second „ (1823—1846)	. 100,448	... 6331	... 6.30
Third „ (1847—1861)	. 113,710	... 3599	... 3.17

But a study of the figures brings out another point. In 1833 the institution was divided into two sections, one of which, the First Clinique, was for the instruction of students, and the other, the Second Clinique, for the training of midwives. Although the two divisions were separated by thin partitions only, and were perfectly alike in all other respects, the death-rate among their respective inmates showed the following differences:

	First or Students' Clinique.		Second or Midwives' Training Wards.
1833-39 . . .	6.22 per cent.	...	5.73 per cent.
1840-46 . . .	9.76 "	...	3.83 "
1847-61 . . .	3.31 "	...	2.92 "

From 1820 to 1822 the death-rate from childbed fever in the Vienna Lying-in Hospital was nearly the same as in many other large maternity institutions. With the year 1823 a rise began, which reached an enormous height; and from 1847 there was a decline, which brought the mortality down nearly, if not altogether, to the level of the first period. Comparing the death-rate in each of the two divisions which had been formed in 1833, we find that from 1833 to 1839 it was nearly the same in both; while from 1840 to 1846 it rose more than fifty per cent. in the first, and fell nearly fifty per cent. in the second; and since 1847 it has fallen in the First Clinique to one third of its previous maximum, thus bringing the death-rate to a uniform mean in the two divisions. The conclusions may be stated as follows: For the period ending with 1822, the sick-rate for puerperal fever in the Vienna Lying-in Hospital depended on influences which were common, more or less, to all similar institutions; after 1823 a new factor came into force, which acted continuously down to 1846, and caused a very material increase in the number of cases and in the mortality; and that factor was shown by Semmelweiss to be indirectly derived from the flourishing state of morbid anatomy in the Vienna School during those very years. "The anatomical vogue," says Semmelweiss, "brought to the professors, assistants, and students many opportunities of coming into contact with dead bodies. It is evident from the cadaveric odour which the hands will retain for some time, that the ordinary mode of washing the hands with soap does not altogether suffice to remove the cadaveric particles clinging to them. In examining women in pregnancy, or during the labour, or in the course of the confinement, the hand tainted with particles from dead bodies comes into contact with the genitals, so that cadaveric matters have the chance of being absorbed and of passing into the vascular system," and thereby of causing septicæmia in the patient. In the following figures we have absolute proof of the correctness of that doctrine: The enormous increase in the mortality (to six per cent. average) which followed the rise of the fashion of post-mortem inspection, lasted from 1823 to 1832, while the institution was undivided; and continued also from 1833 to 1839, after its division into two, or for so long as doctors and midwives were assigned for their instruction to both divisions in equal proportions. From 1840 to 1846 the gross mortality continued about the same; but there is this distinction to be made between it and that of the former period, that in the first division, now reserved for the instruction of practitioners, who were the real agents of conveying the infection, it rose just as much as it fell in the second division now set apart for the training of midwives, who had nothing to do with morbid anatomy. Coming to 1847, we see the death-rate in the first division falling almost

to the level of that in the second; and we discover the explanation in the practice introduced by Semmelweiss, who was then an assistant in the first division. "Let us assume it to be true," he says, "that cadaveric particles clinging to the hand can induce the disease. Then by completely destroying these particles through chemical means, and thereby providing that the generative organs of pregnant or puerperal women shall be touched by the finger merely, and not by particles adhering to it, we ought to be able to prevent the disease—in so far as it is due to dead-house matters introduced in the course of the digital examination." Inspired by this idea, Semmelweiss in 1847 procured the adoption of the rule that teachers and students should wash their hands with chlorinated water before making an examination, or coming in any way into contact with the sexual parts of women in labour or in childbed; and ever since that time the sick-rate and death-rate from puerperal fever in the Lying-in Hospital of Vienna have fallen so materially, that one cannot well hesitate to ascribe the remarkable decrease in the disease to these preventive measures, just as we ascribe its former prevalence in great part to those noxious influences.

In this way Semmelweiss founded the doctrine of the septic nature of puerperal fever. At the same time he laid emphasis on the local character of the infection, by proving that the infective matter was conveyed by the hand of the practitioner or midwife; and thus he provided a basis for the doctrine that child-bed fever is a *traumatic* septic process, to which every puerpera is liable, because the mucosa on the inner surface of the uterus is laid bare as if it were a wounded surface; while most puerperal women are further predisposed on account of more or less considerable laceration of the cervix or of the vagina or of the external parts, caused either by unskilful handling or by the foetus *in transitu*. The conclusion which Semmelweiss drew from these facts was no doubt one-sided, inasmuch as he traced the sepsis exclusively to transmission of the so-called cadaveric poison. It is beyond question that the infecting matter may come from putrefactive changes essentially belonging to the puerperal process itself; and it stands to reason that these will make themselves the more felt the more abundantly they are produced in overcrowded wards, or under whatever circumstances cleanliness and ventilation are rendered most difficult. Moreover, as we may gather from many of the observations above quoted, the infecting substance may come from the pus and ichor that are gene-

rated in various forms of disease. Again, transmission of infection is by no means restricted to the hands of the accoucheur or midwife; experience teaches us that it may take place by means of their clothes, or through instruments (catheters, syringes, &c.), by sponges, bedding, bed-linen and the like, or, lastly, by the air entering with the finger or instrument introduced into the vagina or uterus,¹ perhaps even by means of air entering by aspiration. Furthermore, the observations given above afford a most interesting explanation of the disease spreading, as it is known to spread, from lying-in hospitals to women confined at their own homes; as well as of the so-called "epidemics" of puerperal fever outside lying-in hospitals, which are really a series of infections from case to case, the active media being one or more practitioners or midwives, and the "epidemic" being confined, as has been often noticed, to the practice of one practitioner or one midwife.

§ 170. RELATION TO MICRO-ORGANISMS.

Up to this point in the discussion of the question I have used the current phrase of "septic disease." The term has still a use and meaning, in so far as it denotes the relationship that may be discovered between putrefactive micro-organisms and the morbid cause which underlies the development and propagation of these diseases. In the morbid process itself there can be no thought of "sepsis;" the demonstrated fact that organic bodies (micrococci) are uniformly found in the "septically" affected tissues warrants us in concluding that the products of putrefaction are not in themselves the excitants of disease, but that the micro-organisms stand in some direct relation to the pathogenesis, although for the present we are ignorant of its intimate nature. And that conclusion is all the more justified, since proof has been given experimentally, by artificial infection of animals, that these organisms have a disease-producing action. From the researches of Recklinghausen,² Waldeyer,³

¹ See Spiegelberg, 'Berl. klin. Wochensch.,' 1880, No. 22.

² 'Verhandl. der phys.-med. Gesellsch. zu Würzburg,' 1871, Sitzungsber., xi.

³ 'Archiv für Gynakologie,' 1872, iii, 293.

Heiberg,¹ Birch-Hirschfeld,² Orth,³ Eberth,⁴ Landau,⁵ and others, that proposition holds good for *childbed fever* among other diseases of the kind. We should thus regard puerperal fever as being also a *process of disease due to parasites*, although we should not therefore be justified in speaking, as many observers do, of a specific virus of puerperal fever, just as we speak of the virus of smallpox or of scarlet fever. The facts that have been adduced in detail in the preceding pages serve to show that this virus inheres equally in the products of cadaveric decomposition, and in those of suppurating or ichorous tissues in diseases of all kinds; while the experience of lying-in hospitals teaches us that the same virus can develop in the putrefying membranes and lochia when brought into contact with the air, as well as in the morbid products which arise in the course of childbed fever itself. On the other hand, there are many observations to prove that practitioners, midwives, nurses, infants, and others, who have come into close contact with puerperal-fever patients, may be infected by the latter, such infection leading always to an attack of one or other of the so-called "septic" diseases.

Accordingly, whether we regard puerperal fever from the anatomical and clinical point of view, or from the etiological, it ranges itself with the *traumatic septic diseases*; and it is distinguished from other forms of disease belonging to the same group merely by having a special stamp impressed upon it from the peculiar physiological state of those who are its victims.

§ 171. CAUSAL CONNEXION WITH ERYSIPELAS.

I have one more point in the natural history of childbed fever to discuss, which has a peculiar interest for practice.

¹ In 'Virchow's Archiv,' 1872, lvi, 407, and 'Die puerperalen und pyämischen Processe,' Leipz., 1873.

² 'Archiv der Heilkde,' 1873, xiv, 193.

³ In 'Virchow's Archiv,' 1873, lviii, 437, and 'Arch. für experim. Pathol.,' 1873, i, 81.

⁴ 'Centralbl. für die med. Wissensch.,' 1873, Nr. 8.

⁵ 'Archiv für Gynäkol.,' 1874, vi, 147.

I mean the connexion between that disease and *erysipelas*. Pouteau was the first, so far as I know, to describe puerperal fever as “*erysipelatous inflammation of the peritoneum.*” This is a view of the morbid process arising more out of the nosological fashion of the time, than from any anatomical, clinical, or etiological facts. Among the English accoucheurs of the end of last century, such as Johnstone¹ and Home,² it found a ready acceptance, all the more so that the somewhat frequent coincidence of *erysipelas*—or of what the profession in England called *erysipelas*—with puerperal fever in epidemics gave support to the idea. At length it came to pass that English practitioners hardly doubted the kinship between childbed fever and *erysipelas*; and Nunnely³ went even so far as to say: “This much at least I am sure of, that many questions in medicine, which by common consent are regarded as settled, do not rest upon stronger evidence, if so strong, as that which has been now adduced in favour of the identity of *erysipelas* and puerperal fever.”

The facts upon which the idea of an etiological connexion between *erysipelas* and puerperal fever is based, are the following :

(1) *The coincidence in time and place of the two diseases in epidemic form, both in lying-in institutions and among the population at large.*

Data on this point are furnished by the following writers: Clarke, in his account of the childbed fever of 1787-88 in the lying-in hospitals of London; Gordon, in his narrative of the disease in 1789-92 at the lying-in hospital and in the New Town of Aberdeen; Hey, from observations in Yorkshire, 1808-12; West for Abingdon, 1813-14; Douglas for Dublin, 1819-20; Blackmore for Plymouth, 1831; Sidey for Edinburgh, 1825-26; Elkington and Ingleby for Birmingham, 1833; Beatty for Dublin, 1830-37, and M'Clintock for the same, 1845-46; Ackerley⁴ for London, 1838; and Fox,⁵ from the record of the lying-in charities of London from 1833 to 1858. Similar experiences are recorded by Hodge and Wilson for the lying-in hospital of Philadelphia, where numerous cases of *erysipelas* had been noticed every time that puerperal fever

¹ ‘Observationes de febre puerperali,’ Edinb., 1779.

² ‘Clinical Experiments, Histories and Dissections,’ Lond., 1780, 183.

³ ‘Treatise on the Nature . . . of Erysipelas,’ Lond., 1841, 89.

⁴ ‘Lond. Med. Gaz.,’ 1838, June, 463.

⁵ ‘Transact. of the Obstetr. Soc.,’ 1862, iii, 368.

occurred, and by nearly all the practitioners who wrote on the severe epidemics of erysipelas in America in 1841 and following years.

From the reports by Schäffer and Jespersen on the childbed fever of 1844 in the Sanitary District of Aarhus, it appears that malignant erysipelas had been widely diffused at the very same time; and that was also found to be the case, according to the official report, on the island of Bornholm in 1848. From Germany we have accounts to the same effect for the Vienna Lying-in Hospital from 1819 to 1861;¹ for Neuenhaus 1827-28; for the lying-in institution at Würzburg 1835 and 1846; the Stuttgart Maternity 1849; the Lying-in Hospital of Prague, 1861; and the midwifery wards of the Berlin Charité, 1879.² In the Paris lying-in hospitals many coincidences of the two diseases have been noticed, an enumeration of which down to 1848 is given by Masson.³ The following account, by Pihan-Dufeillay, of the epidemic of puerperal fever in 1861 at the Hôpital St. Louis has a special interest: About the end of January or beginning of February an epidemic of puerperal fever broke out among the patients in the lying-in wards. Notwithstanding the excellent sanitary arrangements of the wards, it quickly assumed so malignant a character that new admissions were stopped and the patients (whether with puerperal fever or free from it) removed to other wards. Into the wards thus emptied were transferred thirty-two women suffering from chronic skin-diseases, among whom many cases of erysipelas soon appeared, some of them being of a very severe type, and one (in a syphilitic subject) ending fatally as rose in the face. Among more recent accounts of the two diseases occurring together in French lying-in hospitals are those relating to the Maternité of Lyons⁴ in 1866, the Hôpital de la Pitié⁵ of Paris in 1868, and the Hôpital St. Antoine⁶ of Paris in 1869.

(2) The familiar fact that *women in labour, attended by doctors or midwives who were suffering themselves from erysipelas or had come into contact with erysipelas patients, have taken puerperal fever.*

A number of instances of this will be found in the series of cases above given (7, 8, 9, 10, 11, 13, 14, 44.), where doctors or midwives carried

¹ "As regards erysipelas," says Späth, "it is impossible to deny that it has some intimate connexion with puerperal fever; although, during the epidemic of the former in Vienna and the vicinity in 1853, the state of health among the patients of the lying-in hospital was good. . . . In 1861, erysipelas had been seen from time to time in the Vienna Obstetrical Clinique for Midwives and Practitioners just before the epidemic of puerperal fever broke out in it."

² See Runge, l. c.

³ 'De la coïncidence des épidémies des fièvres puerpérales et des épidémies d'érysipèle, de l'analogie et de l'identité de ces deux maladies,' Paris, 1849.

⁴ Fonteret, l. c.

⁵ Ref. in 'Gaz. des hôpit.,' 1869, Nr. 33.

⁶ Lorain, l. c.; Quinquaud, l. c., 32.

the infection. Many of the American authorities, such as Hall, Dexter, Holston, Corson, Leasure, Galbraith, and Minor, are emphatic in saying that cases of sickness among puerperæ during the great epidemics of erysipelas occurred mostly in the practice of those who had erysipelas patients and had gone direct from these to their patients in labour or in childbed. The remarkable case recorded by Wegscheider has been given already at p. 457. The slight epidemic of puerperal fever at Reichenbach in 1872 arose in the same way, according to the account by Kraus: the midwife of the place was attacked with erysipelas of the face, and was still in the stage of scaling, when she delivered (Oct. 2) a woman at Reichenbach, who fell ill in the course of two days and died on the day following. During October and November this midwife delivered nine other women, who all took puerperal fever, seven of them dying of it. At that time erysipelas was epidemic in Reichenbach and vicinity; in Reichenbach itself there were eight persons suffering with rose in the face, and in two of these cases the disease had a fatal issue. The following case was communicated by Dr. Freer to Spencer Wells, who quoted it in the discussion on puerperal fever at the Obstetrical Society:¹

“Two years since I was engaged to attend the wife of a clergyman in her first confinement—a very fine healthy lady, aged 26. Upon entering the bedroom, I found a nurse in attendance with an erysipelatous blush and swelling upon the side of the face. Upon inquiry she told me that, two days before, she had been in Liverpool Hospital to have a nasal duct opened. My patient was seized with rigors at the end of thirty hours and died of puerperal fever on the eighth day. The nurse died of rapid erysipelas of the head and neck on the twelfth day.”

(3) The converse fact to (2), that *doctors, midwives, nurses, or other individuals who come into close contact with puerperal-fever patients, suffer from erysipelas remarkably often; also that the newborn infants of mothers with puerperal fever die of erysipelas in an unusually large ratio.*

Of this also there is evidence in several of the observations already given. Lec² mentions a case in which the doctor, the nurse and the child, all took erysipelas from the puerperal fever of the mother. Sidey³ speaks of a case in which five persons in the family of a lady with puerperal fever were attacked with erysipelas a few days after the death of the latter. Kraus concludes his account of the Reichenbach epidemic of childbed fever with the remark that four of the newborn children whose mothers were seriously ill, took erysipelas when a few days old, the inflammation spreading over the whole body and ending fatally in two cases. Squire⁴ gives the following case: Having come

¹ ‘Brit. Med. Journ.,’ 1875, July, p. 105.

² ‘Edinb. Monthly Journ. of Med. Sc.,’ 1847, April, 793.

³ ‘Edinb. Med. and Surg. Journ.,’ 1839, Jan., 92.

⁴ ‘Brit. Med. Journ.,’ 1875, May, 673.

straight from visiting a patient with traumatic erysipelas, he delivered a woman who died on the sixth day; two days later the husband died. Shortly after, another woman, who had come out of the house where the erysipelas patient was, took puerperal fever and died; and then there occurred in rapid succession eight more cases of erysipelas (three of them fatal) among persons who could be shown to have visited either the first erysipelas case or the second puerperal case, having themselves some slight abrasions of the skin.

(4) The fact vouched for by many observers that *childbed fever itself has not unfrequently an erysipelatous character*, if I may so speak; or in other words, that *the disease begins to develop from an erysipelas which mostly arises in the lacerated vaginal mucous membrane*.

I am aware that many of the above observations and many more of the same kind (especially some of those from English sources), which have been adduced to prove the intimate or genetic connexion between childbed fever and erysipelas, do not possess the value of evidence inasmuch as they relate, not to erysipelas, but to diffuse phlegmon of the connective tissues and such-like processes; also that erysipelas neonatorum is in many cases to be taken as not true erysipelas, but as septic phlegmon of the connective tissue. But there will always be a great many cases remaining in which no such reserve is called for; cases where there had undoubtedly been “*érysipéle légitime*” which had given rise to childbed fever just as the infection from a patient with puerperal fever has given rise to the same. Volkmann,¹ who shares the scepticism above referred to, and who is certainly not open to the charge of using the term “erysipelas” in too wide a sense, admits that “when simple erysipelas is abroad, there is no doubt that there are sometimes influences at work which generate puerperal fever, when women newly delivered are exposed to them.” Of the nature of these influences I am unable for the present to form an opinion. I leave it for discussion whether Tillmanns² is right when he says (as other observers also say) that “it may be taken as proved that many forms of so-called ‘puerperal fever’ are true erysipelas which had started in lacerations at the entrance of the genital

¹ In Pitha and Billroth's ‘Handbuch der Chirurgie,’ Bd. i, Abth. ii, A, p. 161.

² L. c., p. 46.

passages ;” or whether Hugenberger¹ is more correct in his view of the relationship of the two diseases, when he says that puerperal fever and erysipelas have one and the same cause, namely, “septic” infection, and are therefore to be regarded as co-ordinate effects. Before we can decide whether this theory can be reconciled with the latest discoveries of Fehleisen,² we shall require to have the researches in that direction continued.

LIST OF WRITERS ON PUERPERAL FEVER.³

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Baart de la Faille, in Nederl. Tijdschr. voor Geneesk., 1854, Novbr. Bang, Selecta Diarii Nosoc. reg. Friederic. Hafn., 1789, ii, 240. Barker, in New York Journ. of Med., 1858, May, 377. Bartholin, Act. med. Havn. Ann. 1671-72. Hafn., 1673, 65. Bartsch, in Oester. med. Jahrbücher. Neueste Folge, x, 123. Bayrhofer, Bemerkungen über das epidemische Kindbettfieber. Frankf. a. M., 1812. Beatty (I), in Dublin Journ. of Med. Sc., viii, 76. Beatty (II), *ibid.*, xii. Baudelocque, Abhandlung über die Bauchfellentzündung der Wöchnerinnen. From the French. Potsd., 1832, 67. Berliner, in Deutsche Klinik, 1855, Nr. 17. Berndt, Klinische Mittheilungen, Heft 3 and 4, 233. Bicker, Raadgeving voor den gemeenen man, etc. Rotterdam, 354. Bidault et Arnoult, in Gaz. méd. de Paris, 1845, Nr. 31, 481. Bischoff, in Abhandl. öster. Aerzte, iv, 107. Blackmore, in Provinc. Med. and Surg. Journ., 1845, Nr. 12, 14—16, 21—26. Botrel, in Archiv. gén. de méd., 1845, April, 416. Bouchut, in Gaz. méd. de Paris, 1844, Nr. 6, 7, 10. Bourdon, in Revue méd., 1841, Juni 348. Bowen, in Western Lancet, 1842, June, Nr. 2. Boysen, Observat. in nosocom. obstetr. de febre puerp. maligna. Hafn., 1792. Bradley, in London Med. and Phys. Journ., xxv, 193. Brennan, Thoughts on Puerp. Fever, etc. Lond., 1814. Bruu, in Séance publ. de la Soc. de Méd. de Toulouse, 1830. Burckhardt, in Allgem. med. Annal., 1802, Correspondenzbl., 177. Burguet, in Journ. de Méd. de Bordeaux, 1853, Avril. Burns, Principles of Midwifery, 8th ed., Lond., 1832 (Germ. ed., p. 576). Busch, in Zeitschr. für Geburtskunde, ii, 637. Butter, Account of the Puerp. Fevers as they appear in Derbyshire. Lond., 1775.

Campbell, Treatise on the Epidemic Puerp. Fever, etc. Edinb., 1822.

¹ ‘Archiv für Gynäkol.,’ 1873, xiii, 387.

² See above, p. 414.

³ The list contains only such writings as have a special interest for epidemiology or are referred to in the table of epidemics.

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CHAPTER XIII.

HOSPITAL GANGRENE.

§ 172. HISTORICAL OUTBREAKS. PRESENT GEOGRAPHICAL RANGE.

The third member of the series of traumatic infective diseases which has an interest for our particular subject is hospital gangrene, a malady that connects in a general way with the morbid conditions discussed in the two preceding chapters, inasmuch as it also has been prevalent in all ages over the whole globe. In the medical writings of antiquity and the middle ages, such as the works of the Hippocratic Collection,¹ of Celsus,² Galen,³ Avicenna,⁴ the School of Salerno, Guido de Cauliaco⁵ and De Vigo,⁶ mention is made,

¹ See 'De locis in homine,' § 29 (ed. Littré, vi, 322), for an account of the ulceration described under the name of "*ἑρπῖον*."

² Lib. v, cap. 28, § 3 (ed. Targa, i, 287), where this form of ulcer is described as "*ἑρπίωμα*," and another form, "*φαγέδινα*" (in other readings "*ἑρπης ἰσθιόμενος*," as in the edition by Almeloveen, Basil., 1748, 319), is mentioned as follows: "vocant Graeci hoc ulcus *φαγέδινα*, quia celeriter serpendo, penetrandoque usque ossa, corpus vorat. Id ulcus inaequale est, coeno simile, inestque multus humor glutinosus (might not this suggest the so-called diphtheritic deposit?), odor intolerabilis, majorque quam pro modo ulceris, inflammatio."

³ In lib. iii, 'De temperamentis,' cap. iii (ed. Kühn, i, 664), and 'Comment. in Hippocratis Aphorismos,' cap. xlv (e. c., xviii, A, 71), malignant destructive ulcerations are spoken of as "*φαγέδινα καὶ ἑρπης ἀναβιβροσκόμενος*;" and in lib. ii, 'De praesagitione ex pulsibus,' cap. i (e. c., ix, 273), "ulcera depascentia" are mentioned, "*ὅυς ἰσθιόμενους Ἱπποκράτης ὠνόμαζεν*."

⁴ Ref. to malignant progressive ulceration in 'Canon,' lib. iv, Fen. iv, Tr. iii, cap. 1 ff. (ed. Venet., 1564, ii, 157).

⁵ 'Chirurgie magna,' Tract. iv, Doctr. i, cap. iii (Lugd., 1572, 233: "Dum ulcus non habet nisi sordem et sanie grossam et viscosam, dicitur sordidum; postquam autem augetur ipsius malitia, ita quod putrefacit et mortificat carnem dimittendo sarra, a qua elevatur fumus foetidus, et cadaverosus, dicitur putridum fraudulentum. Et si ambulat ejus malitia, transit ad esthiomenum et ad hominis mortem."

⁶ 'Chirurgia,' lib. iv, Tract. vii, cap. i (Lugd., 1521, fol. cvii).

under various names, of malignant gangrenous ulcerations, progressing rapidly, causing wide-spread destruction of tissue, and sometimes endangering life; and these may be identified, in part at least, with the disease which afterwards received the name of hospital gangrene. More precise references to this disease are met with in the writings of some of the sixteenth and seventeenth-century surgeons, such as Tagault¹ and Marcus Aurelius Severinus², but more particularly in Paracelsus and Paré.

In the "Grosse Wundartzney" of Paracelsus³ there is the following interesting passage: "Be it further said that in times of pestilence, the pestilence has appeared in wounds, unless there were no ulceration. The onset was with chills and heats, and some died of it speedily, having felt nothing else in all their body; for that which brought it on issued from the wounds. Sometimes, too, it has been noticed that a common croup (*Brüune*) has befallen the people in war, which also had all its signs in the wounds, in suchwise that a thick skin came from them like that which one peels from the tongue, the croup affecting them just as if their wounds and their mouths were the same. . . . Of such accidents there have been untold numbers." In his chapter on gunshot wounds, Paré⁴ says, by way of combating the views of those who attribute to some combustion or poisoning the untoward course which such wounds often take, that it is a matter of corruption or "rotting of wounds," under some pestilential constitution of the air that induces the state of rottenness in them. He specially recalls the observations that he made at the siege of Rouen in 1562, when the air was so tainted that the slightest and scarcely noticeable wounds often took on a malignant character. Relating also to hospital gangrene are the statements about malignant ulcerations which he makes in the section⁵ on "neglected, untidy and filthy ulcerations;" he there speaks of a thick tenacious exuvia, like the coating of the tongue in sick persons, which forms upon the wound, while "a corruption and liquefaction of the soft parts" sets in underneath.

The first thorough study of hospital gangrene is met with

¹ 'De ulcere sordido et putri' in *Instit. chir.*, lib. iii, cap. xvi ('De chirurg. script.,' Tiguri, 1555, fol. 103 b.

² 'De efficaci chirurgia,' lib. ii, cap. lii (Frankf. o. M., 1646, 107), on "Ulcera putrescentia;" also in 'Pyrotechnia chirurg.,' lib. ii, Pars. i, cap. viii (e. c., 264), "de gangraenosis et putridis ulceribus," especially after gunshot-wounds; and ib., cap. x (e. c., 265), "de ulceribus cacoethis et malignis."

³ Buch, i, cap. vi, ed. Strassb., 1618, fol. 7.

⁴ 'Livres de la Chirurgie,' Bk. x, in Offenbach's German edition, Frankf. o. M., 1635, pp. 380-81.

⁵ Ib., Bk. xii, chap. ix, 434.

in the works of Mauquest de la Motte,¹ who wrote in the middle of last century, and who gained his experience of the disease in the Hôtel Dieu of *Paris*; and in the writings of Pouteau² who himself came through an attack of the disease at the Hôtel Dieu of *Lyons* while he was a student, and had many opportunities of observing it there subsequently. Shortly afterwards there appeared the monograph on hospital gangrene by Dussaussoy,³ Pouteau's successor at the Hôtel Dieu of *Lyons*; and almost simultaneously the observations by English surgeons, in particular by Gillespie⁴ and Trotter,⁵ on the endemic and epidemic prevalence of the disease on board English ships cruising in *tropical waters* or stationed in the tropics. The wars that involved almost all Europe in the end of last century and beginning of the present, afforded abundant opportunities for studying hospital gangrene; and from that period we have many valuable memoirs on the disease, including those of Boggie,⁶ Hennen,⁷ Blackadder⁸ and Gerson⁹ for the *Peninsula*; Boyer¹⁰ and Delpech¹¹ for *France*; Menzel,¹² Volpi¹³ and Riberi¹⁴ for *Italy*; Thomson¹⁵ for *Belgium*; Brugmans¹⁶ for *Holland*; and Busch¹⁷ for *Treves*. Experiences collected under similar

¹ 'Abhandl. von der chirurgie,' Germ. transl., Nürnberg, 1762, iii, 267.

² 'Oeuvres posthumes,' Paris, 1783, iii, 246.

³ 'Diss. sur le gangrène des hôpitaux,' Genève, 1786.

⁴ 'Lond. Med. Journ.,' 1785, vi, 373.

⁵ 'Medicina nautica,' Lond., 1797, ii.

⁶ 'Transact. of the Edinb. Med.-Chir. Soc.,' 1828, iii, 1.

⁷ 'London Med. Repository,' 1815, iii, 177.

⁸ 'Observations on the Phagedaena gangraenosa,' Edinb., 1818.

⁹ 'Ueber den Hospitalbrand, u. s. w.,' Hamb., 1817.

¹⁰ 'Traité du malad. chirurg.,' Par., 1818, i, 221.

¹¹ 'Mém. sur la complication des plaies et des ulcères, connue sous le nom de pourriture d'hôpital,' Par., 1815. (Transl. into German, along with the work of Brugmans, by Kieser, Jena, 1816, 85.)

¹² In Hufeland's 'Journ. der Heilkunde,' 1799, viii, Heft 4, 144.

¹³ 'Saggio di osservazioni e di esperienze med.-chir. fatte nello spedale civico di Pavia, Milano, 1814.

¹⁴ 'Sulla cancrena contagiosa o nosocomiale,' &c., Torino, 1820. Also in 'Repert. med.-chir. di Torino,' 1822, 214.

¹⁵ 'Report of Observations made in the Military Hospitals of Belgium,' Lond., 1817.

¹⁶ 'Natuurk. Verhandel. van der Holland. Maatsch. der Weetensch te Harlem,' 1814, Aug., vii, St. 2 (Germ. Transl. by Kieser, Jena, 1816).

¹⁷ In Rust's 'Magaz. der Heilkunde,' 1820, vii, 3.

circumstances were published by Surdun,¹ Lallour,² Tourainne,³ and Bourot⁴ after the *Crimean War* in 1854-55; by Goldsmith,⁵ Pittinos,⁶ Brinton,⁷ Thomson,⁸ Packard,⁹ Kempster,¹⁰ Jones¹¹ and Carpenter¹² after the War of Secession in the *Untied States*; and after the late Austro-Prussian and Franco-German Wars, by Lewandowski¹³ for the military ambulance of *Weissenfels* in 1866, by Ponfick¹⁴ for the hospital of *Heidelberg*, by König¹⁵ for the military hospitals of *Berlin* in 1870-71, by Moty¹⁶ and by Leroy¹⁷ for the military ambulances at *Maubeuge* and *Amiens* in 1870-71; after the Austro-Hungarian and Austro-Italian Wars, by Nagel¹⁸ for the hospitals at *Temesvar*, *Pesth* and *Pressburg*, by Rostolli¹⁹ for the ambulance at *Alessandria* in 1848, and by Demme²⁰ for the military hospital at *Milan* in 1859; and after the Indian Mutiny by Moore²¹ for *Sind*.

Besides these memoirs, we have reports of epidemics of hospital gangrene on board ship, which connect with those already mentioned. They come chiefly from *Indian, Chinese and other tropical waters*, the authorities being Curtis,²² an

¹ 'Revue thérap. du Midi,' 1856, Avril.

² 'De la pourriture d'hôpital en général et de celle observée sur les blessés de l'armée d'Orient,' &c., Par., 1856.

³ 'Mém. de méd. milit.,' 1861, iii, sér. v, 303.

⁴ 'Sur la pourriture d'hôpital,' &c., Strasb., 1858.

⁵ 'Report on Hospital Gangrene,' &c., Louisville, 1863.

⁶ 'Amer. Journ. of Med. Sc.,' 1863, July, 50.

⁷ *Ib.*, 279.

⁸ *Ib.*, 1864, April, 379.

⁹ *Ib.*, 1865, Jan., 114.

¹⁰ *Ib.*, 1866, April, 351.

¹¹ 'New Orleans Journ. of Med.,' 1869, Jan., April.

¹² 'Transact. of the Pennsylvania State Med. Soc.,' 1877, 736; and 'Transact. of the Amer. Med. Assoc.,' 1878, xxix, 237.

¹³ 'De pathol. et therap. gangraenae nosocomialis,' Hal., 1866; also in 'Deutsche Klinik,' 1868, 14, 15.

¹⁴ 'Deutsche Klinik,' 1867, Nr. 20 ff.

¹⁵ In 'Virchow's Archiv,' 1871, lii, 376; and 'Ueber Hospitalbrand,' Leipz., 1872 ('Volkman's Samml. klin. Vortr.,' Nr. 40).

¹⁶ 'Sur une épidémie de pourriture d'hôpital,' Par., 1871.

¹⁷ 'Relat. d'une épidémie de pourriture d'hôpital observ. à Amiens en 1870-71,' Par., 1872.

¹⁸ 'Zeitschr. der Wien., Aertze,' 1852, viii, 116.

¹⁹ 'Annal. univ. di med.,' 1849, Maggio, Giugno.

²⁰ 'Militär-chir. Studien, u. s. w.,' Würzb., 1861.

²¹ 'Lond. Med. Gaz.,' 1846, ii, 1005; 1847, i, 187, 450.

²² 'Account of the Diseases of India,' Edinb., 1807, 211.

anonymous writer,¹ Hutchinson,² Smart,³ de Lajartre⁴ and others.

Lastly, there are very numerous accounts of its occurrence in hospitals in all parts of the world by the following among others: Rust⁵ and Fischer⁶ for the Charité at *Berlin*; Schüller⁷ for *Greifswald*, Fock⁸ for *Magdeburg*, Heine⁹ for *Heidelberg*, Alle¹⁰ and Pitha¹¹ for *Prague*, Groh¹² for *Brandeis*, Coote,¹³ Hawkins¹⁴ and an anonymous writer¹⁵ for hospitals in *London*, Bobilier¹⁶ for *Toulon*, Chambolle¹⁷ for *Dunkirk*, Faure¹⁸ for *Spain*, Rigler¹⁹ for *Constantinople*, Pruner²⁰ for *Egypt*, Baudens²¹ for *Algiers*, Adam²² for *Aden*, Curtis (l. c.), Dunbar,²³ Walker,²⁴ Chevers²⁵ and Sutherland²⁶ for various parts of *India*, Friedel for²⁷ *China*, Wright²⁸ for *Baltimore*, Gillespie (l. c.) for the *West Indies*, and Lallemand²⁹ for *Brazil*.

¹ 'Lond. Med. and Phys. Journ.,' 1810, July 13.

² 'Pract. Observ. in Surgery,' Lond., 1826.

³ 'Lancet,' 1870, October, 462.

⁴ 'Considér. sur l'état sanitaire de la frégatte l'Andromaque pendant sa traversée de France en Chine,' Par., 1866.

⁵ 'Magaz. der Heilkde.' 1833, xl, 539.

⁶ 'Charité-Annalen,' 1865, xiii, Heft 1.

⁷ 'Zeitschr. für Chir.,' 1878, viii, 540.

⁸ 'Deutsche Klinik,' 1856, Nr. 25, 26.

⁹ 'Handb. der Chirurgie,' by Pitha and Billroth, Bd. i, Abth. ii, A. 221 ff.

¹⁰ 'Oest. med. Jahrb.,' Nste. F. iii, 594.

¹¹ 'Prager Vierteljahrschr. für Heilkde.,' 1851, ii, 28.

¹² 'Wien. med. Wochenschr.,' 1858, Nr. 35, 36.

¹³ 'Lond. Med. Gaz.,' 1847, i, 729.

¹⁴ *Ib.*, 1028.

¹⁵ 'St. George's Hosp. Reports,' 1866, i, 363.

¹⁶ 'Mém. de méd. Milit.,' 1854, xiv, 60.

¹⁷ *Ib.*, 1843, liv, 247.

¹⁸ 'Souvenirs du Midi.'

¹⁹ 'Die Türkei und deren Bewohner, &c.,' ii, 85.

²⁰ 'Die Krankheiten des Orients,' 158.

²¹ 'Clinique de plaies d'armes à feu,' Par., 1836.

²² 'Transact. of the Calcutta Med. Soc.,' 1837, iii, 132.

²³ 'Ind. Journ. of Med. Sc.,' 1842, New Ser., i, 630.

²⁴ 'Ind. Annals of Med. Sc.,' 1858, Jan., 83.

²⁵ *Ib.*, 1860, Nov.

²⁶ *Ib.*, 1857, April, 471.

²⁷ 'Beitr. zur Kenntn. des Klimas und der Krankheiten von Ostasien,' Berl., 1863, 135.

²⁸ 'Amer. Journ. of Med. Sc.,' 1832, May, 47.

²⁹ In Casper's 'Wochenschr. für die ges. Heilkde.,' 1845, 481.

The facts contained in these various writings afford evidence that hospital gangrene has been a disease of *all times* and of *every part of the habitable globe*. It has been found both in high latitudes and in low, on the coast and in the interior, at elevated situations and at low-lying places, and on dry soil as much as on wet. Although no climate enjoys any notable immunity from it, there is almost complete unanimity¹ among such of the English and French naval and military surgeons as had been for some time in the tropical parts of Asia, Africa and America, that those are the regions most scourged by hospital gangrene. The "foul sloughing ulcers" of the English surgeons in India, the Red Sea ports, the West Coast of Africa, Guiana, &c., and the "phagédénisme tropical" of the French, observed as they have mostly been in hospitals and prisons, take a prominent place among the diseases of the tropics; and in that group of diseases, of which I shall have something more to say when I come to speak of diseases of the skin, hospital gangrene plays the principal part.

§ 173. DEPENDENCE ON CLIMATE AND WEATHER.

It is impossible to decide with certainty to what extent the relative frequency of hospital gangrene in the tropics is dependent on certain factors in the hygiene, to what extent it is to be explained by meteorological influences,—by high temperature and extreme moisture, or by the sudden changes of weather which are so acutely felt in the tropics—and how these influences bear upon the production of the disease. Even with the aid of comparative experiences on the same matter in higher latitudes, we can arrive at no definite conclusion. It is an undoubted fact that hospital gangrene may occur at any season of the year and in all weathers; but that

¹ Moinet ('De l'influence des climats chauds sur le traumatisme chez l'Européen,' Montp., 1866) is almost alone in his assertion that hospital gangrene is much more rarely seen in the colonies than in Europe. He contradicts himself, moreover, when he adds that the malady occurs there very often, and in severe forms, when the hospitals are crowded, at a time of great epidemic sickness, or when the hardships of living are felt in expeditions to distant places.

fact is far from warranting Fischer and others in peremptorily denying the influence of the weather on the origination and prevalence of the disease. The high or low level of the thermometer is probably without significance in itself for the development and diffusion of hospital gangrene; at least, there are as many observers to speak to very low temperature at the time of the outbreak, as there are to attest that the weather was particularly hot.

On the other hand there is a certain amount of agreement among the authorities as to the influence of extreme fluctuations of temperature on the pathogenesis, and so far also of a higher degree of atmospheric moisture dependent thereon; or, in other words, of that kind of weather which is especially characteristic of the changes of season in the tropics. This is the position taken up by many of the Anglo-Indian physicians.

Thus Curtis points to the prevalence of the disease in Madras in October, 1782, during the monsoon, and its extinction on the setting in of dry and cool weather. Adam tells us that hospital gangrene broke out at Hussingabad (Kandeish) among the ill-clad and unprotected sepoys in October, 1818, when a cold and damp wind began to blow from the north-east, although only a few cases occurred among the rest of the troops. Chevers says that in Bengal and the N. W. Provinces the wet season of the year is the proper season of hospital gangrene. The epidemics observed by Moore and Dunbar happened in the rainy or cold season. Dussaussoy had previously learned from his Lyons observations that hospital gangrene was most frequent with high temperature and a long prevalence of moist south winds; and a similar conclusion was come to by Boyer, Boggie, Larrey¹ and Hennen from their experiences in the Peninsular War. Groh lays most stress, with reference to Hungary and Italy, on variations in the amount of atmospheric moisture associated with extreme fluctuations of temperature. Gerson and Bobilier, and the United States' surgeons in the War of Secession, found cold and damp weather to be favorable to the development of the malady.

¹ 'Mémoires de Chirurgie militaire' (Germ. ed., i, 440).

§ 174. OFTEN ASSOCIATED WITH AN OVERCROWDED AND UN-
WHOLESOME STATE OF THE HOSPITALS, BUT NOT ALWAYS SO.

Not less considerable than these differences of opinion as to the influence of the weather, are those relating to the significance of *unhygienic conditions*, such as overcrowding of the wounded within doors, deficient ventilation and cleansing of the crowded apartments, in short, the whole of the noxious influences, chiefly met with in hospitals, which it is the practice to group together under the name of "hospitalism." In the first place, it has to be said that the disease has been by no means exclusively confined to hospitals, as the name implies; nor is it to be designated a hospital disease pre-eminently. There is a not inconsiderable series of observations serving to prove, not only that the disease had been observed outside the hospitals at a time when it was epidemic within them, but that it had even originated outside the hospitals, the first cases in these having been such as were brought in. That such might be the case had been shown by Moreau and Burdin,¹ from their experiences in the Napoleonic Wars; and the same thing was observed by Moore in the Sind war in 1845-46. In the epidemic of 1831-32 at the hospital of Prague, the first case, according to Allé, came from the town; and the same was observed on a still larger scale, as Pitha tells us, in the epidemic of 1850. When hospital gangrene was epidemic in the Berlin Charité in 1856, and again in 1864-65, there were cases at the same time in the town, having no connexion with those in the hospital (Fock, Fischer). The same circumstance was noted at Heidelberg,² in 1866-68, at Baltimore in 1830-32 (Wright), and in London, where many cases of hospital gangrene were admitted for treatment into St. Bartholomew's and St. George's Hospitals in 1846 (Hawkins), and into St. George's again in 1869.³

Another argument against the influence of hospitalism in

¹ In Sedillot, 'Journ. de méd.,' An. v, i, 353.

² Herne, l. c., p. 267.

³ Leigh, 'Lancet,' 1869, Oct. 16.

causing the development and diffusion of hospital gangrene has been discovered in the fact that the disease has not unfrequently appeared in newly-built, clean, airy, and far from crowded wards ; while filthy and ill-ventilated wards, closely packed with surgical cases, have escaped. Boggie, confirming Bell,¹ pointed out that the malady had been prevalent during the war in the Peninsula in thoroughly clean and well-ventilated hospitals. Coote says that when the disease appeared at St. Bartholomew's Hospital in 1846, the wards were well aired, well lighted, clean, and in no wise crowded ; and in the epidemic of 1863-65 at St. George's Hospital, hospital gangrene was most severe in one of the best wards, while the surgical cases in two of the most unwholesome wards were free from it ; even the most thorough ventilation of the wards was of no avail. In the military hospital at Antwerp the disease made progress, notwithstanding ventilation and the most scrupulous cleanliness.² Speaking of the epidemic at Prague in 1831-32, Allé says : " The wards were neither overcrowded, nor were there many patients with extensive and foul ulcerations. The keeping of the air in the wards pure and the bandages clean was attended to, as it always was ; in fact, after the hospital gangrene showed itself, the upper casements of two windows in each ward were left open day and night, even in the most intense cold, a double quantity of fuel being served out to make up for the heat lost." It was the same when the disease broke out again in that hospital in 1850 ; the number of patients was absolutely small, particularly the number of operation cases, being less than in the previous three years ; while there was no difference as regards ventilation and cleanliness.³ Wright informs us that when the disease broke out in 1830 in the hospital at Baltimore, the wards were emptied as far as possible and everything done to ensure the utmost cleanliness and adequate ventilation ; but the malady continued to progress. Referring to the frightful epidemics of hospital gangrene that occurred year after year in the central prison at Agra, Walker says : " hygienic

¹ 'Principles of Surgery,' Edin., 1801, i, 108.

² Clements, 'Würzb. med. Zeitschr.,' 1863, iv, Heft v.

³ Pitha, l. c.

measures on a grand scale, resulting in the most scrupulous cleanliness, were found of no avail." Chevers quotes a statement by Brougham that, during the siege of Delhi, although the hospitals were crowded, not a single case of hospital gangrene occurred in them. In the epidemic of 1851 at Brandeis, the disease occurred in the airy and sparsely occupied rooms of the Imperial Schloss which had been converted into a hospital. Under equally favorable conditions of place, Marmy saw hospital gangrene in a villa on the Bosphorus belonging to the Viceroy of Egypt. Fischer says of the epidemic in 1864-65 at the Berlin Charité, that it began in a room which had stood empty the whole summer, and had then been thoroughly cleaned out and whitewashed afresh. On the other hand, in two low-roofed wards on the third floor, somewhat crowded and ill ventilated, not a single case of hospital gangrene was seen. During the Secession War in the United States, hospital gangrene was introduced from Richmond into the hospital at Annapolis, although the arrangements in the latter, as Pittinos tells us, were in every respect excellent. Packard's experiences in the military hospital of Philadelphia led him to the same opinion; but he is careful to add that overcrowding, filth, and other sanitary defects were a material help to the disease spreading. In the French military hospital at Maubeuge, according to Moty, there was not a single case of hospital gangrene during the autumn of 1870, despite overcrowding, want of cleanliness, and lack of surgical aid; it was not until December that the disease broke out, by which time the sanitary condition of things had been very much improved.

Noteworthy as these and many other observations like them are, there is a not less considerable weight of evidence on the other side, which does not permit us to ignore the influence of the above-mentioned defects of hygiene on the origination and diffusion of the malady. As early as the end of the sixteenth century (1597) the surgeons of the Hôtel Dieu of Paris had occasion to notice that hospital gangrene occurred almost uniformly in a ward set apart for the reception of injured persons, which was known by the name of "Rang noir," and in which the beds were close together and hung with curtains, so that there could be no

proper ventilation.¹ The same tainting of the hospital atmosphere is assigned by Dussaussoy as the most material cause of the endemic prevalence of hospital gangrene in the Hôtel Dieu of Lyons. Bobilier found the reason of the disease being endemic in the military hospital of Toulon to be the situation of the edifice in a narrow and ill-ventilated street with high houses all around, where the air was loaded with putrid products of decomposition from the badly constructed latrines and from the graveyards, which were in badly chosen situations. The endemic of hospital gangrene in the hospital of Dunkirk could be traced, as Chamboll tells us, to water standing in an adjoining hollow; when that was removed the endemic came to an end. "So long," says Rigler, "as the medical institutions of Constantinople fell short of the admitted requirements in point of situation, airiness, internal arrangements, and administration, we saw cases of hospital gangrene; but it disappeared when the inconvenient, low, damp and dark buildings were abandoned and replaced by hospitals better situated, dry, well-lighted and well-aired." Under the same circumstances as Rigler in Constantinople, Pruner saw the disease prevalent in the hospitals of Alexandria, Abou-Zabel, and other places in Egypt. It stands to reason that the corresponding observations in times of war should have been on a much larger scale. Wenzel states from his experience of the Italian military ambulances that crowding of buildings, want of ventilation, filth in the wards, and the like, very materially helped to generate the disease. Brugmans concluded from his observations, collected in the military ambulances of the Netherlands, that "the disease was first generated in low, dull, damp and ill-ventilated apartments, and was more difficult to combat in these than elsewhere." Hennen took the same view in the case of the military hospitals in Spain. Other evidence in this direction is given in the account by Rostolli of the epidemic of hospital gangrene from 1848 to 1849 in the overcrowded military hospital at Alessandria, in that by Albespie relating to Genoa, as well as by Bourot, Lallour, Marmy, and other French army surgeons in the military hospitals in the Crimea and at

¹ Foderé, 'Leçons sur les épidémies,' &c., Par., 1824, iii, 495.

Constantinople during the campaign in the East; which latter experiences are summed up by Tourraine in these words: "L'encombrement des hôpitaux est, sans contredit, la cause la plus commune, la cause occasionelle de la pourriture d'hôpital."

In nowise different are the accounts given by several United States' surgeons, such as Goldsmith, Thomson, Packard, and Kempster, concerning the state of matters in the military hospitals during the War of Secession. And not less telling are the observations on the endemic or epidemic prevalence of hospital gangrene on board ships of war. Referring to the frightful havoc that the disease used to make in the English navy,¹ one of the authorities writes: "In dirty ships (a comprehensive term, including the actual impurities that may be suffered to accumulate in every part, the want of ventilation, and inattention of the crew to personal cleanliness) the disease was found most to prevail."

The French surgeons had the same experience during the Crimean war on board transports crowded with the wounded; so also the United States' surgeons during the War of Secession. Of special interest in this respect is the report of Brinton, who was sent on a visit of inspection to the hospitals of the Confederate army in order to inquire into hospital gangrene:

¹ Hutchinson ('Pract. Observ. in Surgery,' Lond., 1826) says that at the time when he first entered the naval service (beginning of this century) cases of hospital gangrene were of the commonest occurrence in the fleet; so much so that not unfrequently the greater number of the ships of a squadron were obliged to return to port on account of it. In a paper written about that time ('Lond. Med. and Phys. Journ.,' 1810, July, p. 13) the following passage occurs: "For some years a species of ulcer, called the ship-, or contagious malignant ulcer, had afflicted the British navy to an extent that was extremely alarming Some idea may be formed of the ravages occasioned by this malady when it is stated that in 1804 six hundred and twenty-one patients labouring under it were admitted into a single hospital at Plymouth. In 1805, Sir Edward Pellew, Commander-in-chief in India, stated to the Admiralty that so destructive was the ship-ulcer, and so intractable under every kind of medical and chirurgical treatment, that an apprehension was entertained of some of his ships being depopulated. In 1804 and 1805 it had made destructive inroads upon the health of the fleets off Brest and Ferrol; but the greatest fatality occurred in 1806 on board the "Salvador del Mundo," then a receiving ship at the port of Plymouth. When Dr. Andrew Baird . . . visited the fleet off Brest and the squadron at Ferrol, he found in many ships the progress of this disease truly awful."

"The disease occurred almost always in patients who had been wounded at the battle of Murrfreesboro', and who had been retained in crowded hospitals for some time previous to their transportation to Louisville. I am informed by Surgeon Thurston, U.S.A., Medical Director of the Nashville Hospitals, that no one upon whom the gangrene had already appeared was ever sent from Nashville, and yet many were so infected when admitted to the Louisville hospitals. The development of the disease on the route seems to have been owing to the fact that the transportation of the wounded was effected by means of crowded and ill-ventilated boats, and the trip by the Cumberland and Ohio rivers frequently occupied several days. During this time these patients, who had already undergone much suffering, were exposed to all the influences most apt to engender this disease. In contrast with this fact it was found that as soon as the Louisville and Nashville railroad was opened, so that the wounded could be conveyed from city to city in one day, all importation of gangrenous sores into Louisville ceased. The development of hospital gangrene during the boat transportation is a noticeable fact, and is strikingly analogous with the same phenomena observed among our paroled wounded prisoners from Richmond, received into the Annapolis General Hospital some months since."

It is just this preponderant, if not absolute, association of hospital gangrene with hospitals, prisons, ships, and such like self-contained places, where every kind of sanitary neglect has often been rampant, as well as the very notable remission of the disease since those nuisances have been abolished, or at any rate considerably lessened by the application of rational principles of hygiene—it is just these things that serve, in my view, to prove incontestably that the development and diffusion of the disease are, if not absolutely dependent on such injurious influences, yet very materially favoured by them. It would be completely inverting the natural order if we were to pass by these latter facts unnoticed, or to deny their importance, because of a too close attention to those other observations which tend to show that hospital gangrene may develop under conditions that are the reverse of these, or that it does not always develop in surgical wards although such insanitary conditions are present. The mistake made by the observers who take up this point of view in their estimate of the facts, partly arises from their hazy notions of the importance of hygienic defects for the breeding of infective diseases in general, and partly from their anxiety to have hospital gan-

grene characterised as an "epidemic contagious disease," as if there were such a thing as the epidemic of a disease by itself, or as if by this way of speaking they gained anything better than a phrase with no meaning of its own. But, moreover, that term must lose all meaning whatsoever when we see how, in order to prove an "epidemic" origin for hospital gangrene, they have mixed it up with various other infective diseases prevalent at the same time, such as cholera, typhoid, typhus, and diphtheria, whereby the homogeneous character of the disease is completely lost.

§ 175. A COMMUNICABLE DISEASE DUE TO A MORBID POISON.

There is no doubt that hospital gangrene is due to a peculiar morbid cause, to a *specific morbid poison*, for the action of which on the body it is necessary that there should previously have been a wound (König), and which induces in the first instance a local infection at the seat of injury, as in erysipelas and puerperal fever. Whether the above-mentioned influences of weather and insanitary conditions are favorable to the development or the reproduction of this morbid poison, or whether they merely increase the predisposition of the individual to take the sickness (possibly by changing the character of the wound), can hardly be decided at present, the less so that there is nothing certainly known of the nature of the poison, although it is probably of the organised or parasitic sort.¹

The *communicability* or *contagiousness* of the disease, which has been called in question by Allé, Thompson, and the writer on the epidemic at St. George's Hospital (London) in 1863-65, has been demonstrated by means of experimental transmission of the infection to animals (Fischer), and proved still better by means of intentional (Ollivier) or unintentional infection of small abrasions in medical attendants and nurses.² It has been shown that the virus of hospital gangrene can

¹ Hüter ('Centralbl. für die med. Wissensch.,' 1868, No. 12, and 'Zeitschr. für Chirurgie,' 1872, i, 91), in cases of "gangrena diphtheritica," has found spores and fungi (micrococci) in the blood and in the gangrenous tissues.

² See the collection of facts on this point made by Heine, l. c., p. 287.

be transmitted by bandages, instruments, or the surgeon's or nurse's hands soiled by the gangrenous discharge, these being the media of carrying it in a fixed state. It is still a question whether it can be transmitted by currents of air.

§ 176. RELATION TO ERYSIPELAS, PUERPERAL FEVER, AND DIPHTHERIA.

As to the relation between *hospital gangrene and the other infective traumatic diseases of the same group, erysipelas and puerperal fever*, we can say no more with certainty than that the production of them all alike is under the common influence of certain external factors, and that accordingly they now and then coincide both in place and time. But I should consider it altogether unjustifiable to conclude therefrom that the processes are identical, or that the specific cause underlying them all is the same. In fact Pitha's view of the analogy between puerperal fever and hospital gangrene being so close that "it is not going too far to declare unreservedly for their identity" (an opinion which he bases on the coincidence of the two diseases in time), has found very few adherents as yet. In like manner as regards erysipelas and hospital gangrene most observers, such as Goldsmith, Brinton, Ribéri, Ponfick, Fischer, Schüller and Carpenter, content themselves with making good the fact that the two diseases sometimes occur epidemically side by side, without venturing to regard them as identical morbid processes.¹

A very lively controversy has arisen in recent times around the question of the *relation of hospital gangrene to diphtheria*.

¹ In controverting Hüter's theory of monads, König very justly asks, "What is it that leads the monads at one time to attack wounds in epidemic fashion, and in them to spend their activity, causing a diphtheritis of wounds; and another time to leave the surfaces of wounds at peace, to offer no interference with their healing, but to wander into the skin and there cause erysipelas; or, in a third case, to choose the subcutaneous connective tissue as the scene of their labours, and there to set up diphtheritic phlegmon? How is it that the contagion of gangrene is so transmitted as to produce only gangrene, and that of the rose so as to set up merely the rose again in the person infected? Why is it that we can discover no other relationship between rose and hospital gangrene than that rose may supervene on a wound attacked by gangrene, but neither more readily nor less readily than on wounds not infected at all?"

One idea is that of an anatomical, clinical, and etiological community in the two diseases; and that idea has at length come to imply that "hospital gangrene is nothing else than diphtheria of wounds." Indications of the so-called diphtheritic character of the wounds in hospital gangrene occur as far back as Paracelsus and Paré;¹ in later times this view was developed by Ollivier,² Robert,³ and Nagel, and quite recently it has found its more important defenders in Heine⁴ and Carpenter.⁵ I reserve my more special discussion of this question until the chapter on angina maligna (diphtheria of the throat). I will only remark here that the theory rests upon a vague notion of what is "diphtherial" (or, to use a barbarism, "diphtheritic"); and that there is not the slightest ground, from the geographical, historical, or etiological point of view, as I shall show at the proper place, for identifying the diseases. They have nothing more in common than the anatomical condition up to a certain point.

¹ See p. 477.

² 'Traité expérimental du typhus traumatique, gangrène ou pourriture des hôpitaux,' Paris, 1822. I have no knowledge of this work except from extracts. It is impossible that the word "diphtheritic" could have been used by Ollivier, as it was first introduced into medical terminology by Bretonneau in 1827.

³ 'Bull. gén. de thérap.,' 1847, Juli; 'Gaz. des hôpit.,' 1847, No. 85.

⁴ L. c., p. 207.

⁵ "Hospital gangrene and diphtheria are precisely similar" ('Transact. of Amer. Med. Assoc.,' 1818, xxix, 245.

CHAPTER XIV.

CHLOROSIS AND ANÆMIA.

§ 177. VAGUE STATEMENTS ON THE CONNEXION OF ANÆMIA WITH CIVILISATION.

“Poverty of the blood, green sickness, and allied conditions, as the prevailing physical characteristics of our age”—so runs the title of an address¹ given by Politzer in 1856, at the thirty-second annual meeting of the *Deutsche Naturforscher-Versammlung*. In this address he expressed with much eloquence the old complaints of Hufeland and others as to the “physical degradation of the human race,” the “degeneration of mankind to simulacra,” and so forth; and as the characteristic mark of the degeneration he named the group of morbid conditions which I have placed at the head of this chapter under the general term “anæmia.” Evidence that his complaint was in any degree well-founded, the orator did not adduce; and if we follow the only trustworthy road, that of statistics, there is no such evidence to be had. After we have resolved that comprehensive term “poverty of the blood” into its elements, we shall find infinite difficulties in the way of arriving at a safe opinion whether any one of them has, as a matter of fact, undergone so considerable an increase in the extent of its prevalence in modern or recent times as to warrant us in speaking of it as characteristic of the period.

It is not to be denied that progressive cultivation and the refinements of civilised life have introduced, and are introducing every day many things into our mode of living which have a deteriorating influence upon the race, and are favor-

¹ Reprinted in the ‘*Zeitschr. der Wiener Aerzte*,’ 1857, Heft ii, 62.

able to anæmia in particular ; and therein we may find an *a priori* justification of the assertions made. On the other hand, it should not be forgotten that the progress of culture and civilisation has obviated or overcome many sins of omission or commission in the lives of former generations, which were not less injurious to nutrition and blood-making in man's body. And thus the title of the address quoted at the beginning of this paragraph must remain a mere idea, resting on altogether general and very vague impressions—an idea which has no more definite value than the maxim of the melancholy philosopher, that “the world is always growing worse.”

Without entering upon this question, to which it seems to me there can be no answer, I shall take as the subject of a more modest inquiry two or three forms of anæmia, which are of especial interest from the point of view of their geographical distribution.

I. *Chlorosis*.

§ 178. REFERENCES IN ANCIENT AND MEDIÆVAL WRITINGS.

To the practitioners of antiquity and the middle ages, chlorosis, like chronic disorders of nutrition in general, was quite unknown as a peculiar and individual form of morbid process. There are, however, here and there in the writings¹ of the Greek physicians references to pallor (ἀχροΐν) or a yellow tint (χλωρὰ χρώματα) of the skin as symptoms of illness, which may be supposed, in some instances at least, to point to chlorosis. But we find more definite evidence that the disease was not infrequent in those times, in the

¹ Hippocrates, ‘Praenot. coac.’ sect. ii, § 333, ed. Littré, v, 656, and ‘Prorrh.’ lib. ii, § 31, e. c., ix, 64 ; also Galen, in ‘Hipp. Prorrh.’ lib. i, comment. iii, cap. ci, ed. Kühn, xvi, 727 ; and in ‘Hipp. libr. de alimento,’ comment. iii, cap. xvii, e. c., xv, 327, where we have a more precise indication of chlorosis and anaemia ex metrorrhagia : “ὥς μὲν οὖν πολλὰ ἔπεται συμπτώματα ταῖς ἐπισχέσει τῆς ἐμμήνου καθαρσέως, οὕτω καὶ ἀμέτροις κενώσεσιν ἐπιγίνεται τάδε ἄχρoιαι καὶ ποδῶν οἰδημα καὶ ὄλον νποιδον τὸ σῶμα.” The word “χλωρώσις” does not occur in Hippocrates, or in any of the subsequent Greek writers on medicine.

fact that Oribasius, Aetius, Rhazes, Avicenna, the Salernians¹ and others dwell very particularly upon "general weakness of body, trouble in breathing, and paleness of the skin" as symptoms of a disorder of menstruation (*suppressio mēstruorum*) which did not depend on disease in the uterus. In the sixteenth century the malady was well known under various names which described its characters well; such as "*febris amatoria*,"² "*foedus virginum color*,"³ "*morbus virgineus*,"⁴ and "*febris flava*."⁵ The term "*chlorosis*," in the sense in which it is now generally employed, I find occurring first in Sennert,⁶ and in several of the dissertations⁷ written at Basel in the beginning of the seventeenth century. It forms one of the stock subjects in the compendiums of medicine and treatises on diseases of women belonging to that and subsequent periods, the first thorough handling of the matter being in an essay by Friedrich Hoffmann,⁸ in which use is made of the term "*Bleichsucht*," still employed in German colloquial speech. This essay was the source of numerous writings on chlorosis throughout the eighteenth century.

§ 179. PRESENT DISTRIBUTION: REMARKABLE EXTENSION IN SWEDEN OF LATE.

There can be no doubt, then, that chlorosis has been among the subjects of medical observation at all times. But the frequency of the disease at various periods of the past history as compared with its present frequency is a question that we

¹ See "*De egritudinum curatione tract.*," in De Renzi's '*Collectea Salernitana*,' Napol., 1853, ii, 331.

² In Lange, '*Epist. med.*,' lib. i, epist. 21.

³ In Ballonius, '*De virginum et mulierum morbis*,' cap. vii, Opp. Genev., 1762, iv, 66.

⁴ In Rodericus a Castro, '*De univ. mulierum med.*,' lib. ii, cap. v.

⁵ In Mercado, '*De morbis mulierum*,' lib. ii, cap. vi.

⁶ '*Pract. med.*,' lib. iv, tract. ii, sect. iii, cap. ii, Wittbg., 1760, p. 214. The opinion referred to in this passage, that the word *χλωρώσις* had been used by Hippocrates, rests, as we have seen, on an error.

⁷ Flach, '*Diss. de chlorosi, s. morbo virginum*,' Basil., 1621.

⁸ '*De genuina chlorosis indole, origine et curatione*,' Hal., 1731, in Opp. Genev., 1748, suppl. ii, pars. ii, 389.

have no answer for. Even in our inquiry into its *present geographical distribution*, it is impossible to arrive at a definite idea of the relative amount of the sickness at various parts of the world, owing to the data of authorities being fragmentary as well as extremely vague.

In Central and Southern Europe, including *Great Britain and Ireland, Holland, Belgium, Germany, France, Italy*¹ and *Turkey*,² as well as in the countries of *America* within the temperate zone, the malady appears to be common everywhere. From the northern regions of both hemispheres we have accounts of its very considerable prevalence in Miquelon (*Newfoundland*)³ and in *Iceland* and the *Faröe Islands*.⁴

Schleisner's⁵ statement that the malady is very rarely seen in Iceland, is contradicted by Hjaltelin⁶ and Finsen⁷ in the most decided terms. Hjaltelin found that 5 per cent. of all his patients there had chlorosis, and 11.1 per cent. of all the women patients. Finsen treated 316 cases of chlorosis in ten years in a district with 10,000 inhabitants, and he accordingly rates the amount of the sickness very high.

In the annual health reports of *Norway*,⁸ there is mention of the exceedingly frequent occurrence of chlorosis for every district of the country, from Christiansand up to Tromsøe, in the interior as well as on the coast; and in some districts it is accorded one of the first places among chronic maladies. These data are fully borne out in the most recent account of chlorosis in Norway by Axel Lund.⁹ Of recent years the disease has played a not less prominent, possibly a more

¹ See De Renzi ('Topogr. e. statist. med. della città di Napoli,' Nap., 1845, p. 321), on the commonness of chlorosis in Southern Italy.

² Rigler ('Die Türkei und deren Bewohner,' ii, 412), in like manner, speaks of the very considerable amount of the sickness in Turkey and adjoining countries of the Levant.

³ Gras ('Quelques mots sur Miquelon,' Montp., 1866, p. 35) says: "La chlorose domine toute la pathologie de la jeune miquelonnaise."

⁴ Martius, 'Revue méd.,' 1844, Fevr. In Greenland, according to Lange ('Grönland's Sygdomsforhold,' Kjöbenh., 1864, p. 28), chlorosis would appear to be very uncommon.

⁵ 'Island undersögt fra et laegevidensk synspunkt,' Kjöbenh., 1849, 4.

⁶ 'Sundhedskoll. Forhandl. for aaret 1859,' 434.

⁷ 'Jagttagelser angaaende Sygdomsforholdene i Island,' Kjöbenh., 1874, 60.

⁸ 'Beretninger om Sundhedstilstanden i Norge.'

⁹ 'Nord. med. Arkiv,' 1875, vii, Nr. 1.

prominent part in the sickness of *Sweden*. "Within the last twenty or twenty-five years," says Huss,¹ writing in 1851, "a form of disease that used to be rarely seen, has come to be more and more domesticated among grown-up people in Sweden. I mean the green sickness. At an earlier period the malady had occurred in this country among those classes whose children, the girls in particular, had been more delicately brought up, as well as among the poorer classes in the towns; but until thirty years ago or less, it was quite unknown among the inhabitants of country districts. . .

. . The more general diffusion of the sickness has not been a sudden thing with us, but gradual; neither has it been to the same extent in every region of the country, being rather common in some, merely sporadic as yet in others, while in still others it has not been observed at all hitherto. Thus in Norbotten, chlorosis is still unknown as a disease of the people, being rare even in the towns. The most northern point where it can be definitely shown to have become truly endemic is the parish of Skellefteå in the province of Westerbotten. In Angermanland, Medelpad and Jemtland, it was first seen about ten years ago [written in 1851]; and since then it has become more general every year. The same is true of Helsingland and Gestrikland, where chlorosis was quite unknown twenty or thirty years ago; also of the southern parts of Dalarne and Westmanland, whence we are told by Altin that preparations of iron were dispensed by the chemist at Westerås from 1845-50 to the amount of three hundred and seventy per cent. more than in the five years preceding, and by Pallin, that old people had often assured him that no one had ever heard of green sickness in that part of the country twenty years ago. In Upland, Södermanland and Nerike the first appearance of the disease dates from the fourth or fifth decade of the century; and the importance that it has now attained in some districts of these provinces will appear from the statement of Klintberg, that in his practice (in the province of Nerike) only a few of the young women between fourteen and twenty-one years of age were free from chlorosis. On the other hand, it is noteworthy that there is exemption

¹ 'Om Sverges endem. Sjukdomar,' Stockh., 1852, 96.

from the disease in the inland districts of Wermland and Dahlsland, as well as in the coast district of Bohuslän. By far the greatest extension of the malady, both in town and country, appears to have taken place in Westergothland, Ostergothland, Halland, Schonen and Blekinge; and in those regions also it is within the last twenty or thirty years that chlorosis has become common. But in Småland it is less general, and on Gottland it is seldom seen. All the medical accounts [that have been used by Huss for this summary] are agreed in saying that chlorosis is to be regarded as a disease newly arisen among the rural population of Sweden, having first appeared twenty years ago in some of the districts, and in others fifteen, ten or eight years ago, and having for the most part become general in a district soon after it had showed itself at one point."

These statements as to the ever encroaching area and the increasing number of cases of chlorosis in Sweden are fully confirmed in the medico-topographical accounts of Swedish practitioners, published in the official sanitary reports for that country from 1852 to 1871;¹ so that chlorosis may be described as a truly endemic malady of Sweden.

I find only a few accounts of the occurrence of chlorosis in tropical and subtropical countries, and these are partly contradictory and therefore not altogether trustworthy. On the coast of *Mexico*, according to Heinemann's² experiences during a six years' residence in Vera Cruz, it is very common; so much so that it is spoken of as "one of the most general diseases of the Vera Cruzan youth, especially those of the female sex." In the *West Indies*³ it would appear to be almost as common among women as it is in European countries. According to Waddel⁴ there is a good deal of it in *Brazil*; but Wucherer⁵ says that it was rarely to be seen in Bahia. For the *West Coast of Africa* Chassaniol⁶

¹ 'Sundhedskollegii Berättelse.' Unfortunately in these reports since 1871 the medico-topographical accounts have ceased.

² 'Virchow's Archiv,' 1873, lviii, 178.

³ See Savarésy, 'De la fièvre jaune,' &c., Napl., 1809, 88, and Ruz, 'Arch. de méd. nav.,' 1849, Novbr., 346.

⁴ In Castelnau, 'Expedition,' II, 38; but it is doubtful whether there is not some confusion here with other forms of anæmia.

⁵ 'Arch. für klin. Med.,' 1872, x, 379.

⁶ 'Arch. de méd. nav.,' 1865, Mai, 508.

observes that it is less common among the negresses than it is among women in Europe; and the opinion of Frank¹ and Pruner² as regards *Egypt* is the same. On the other hand, according to French authorities, it is very common in *Algiers* among women of Moorish descent. That it is a comparatively frequent malady in *India* we learn from Huillet's³ account for Pondicherry, and from Curran's⁴ for the southern slopes of the Himalaya. In the *East Indies* the cases of chlorosis that Heymann⁵ has seen have been almost entirely in European young women. In the maladies of women in *Cochin China*, according to Beauflis,⁶ chlorosis plays a prominent part along with other forms of anæmia; and that it is also true for *China*, according to Dudgeon⁷, and for *Japan*, according to Wernich.⁸

§ 180. INDEPENDENT OF CLIMATE, SOIL, AND RACE.

Although our information on the history and geographical distribution of chlorosis is fragmentary, there is enough of it to warrant the conclusion that the malady has been observed at all times, that its area of diffusion embraces a large part, perhaps the larger part of the globe, and that no kind of *climate* or *soil* precludes it. We ought not, perhaps, to dismiss absolutely the idea that *extremes of temperature*, such as are found in the highest and lowest latitudes, are especially likely to generate the disease. In favour of that idea is the considerable amount of the sickness in the polar and the temperate cold zones, and its relatively common occurrence in the tropics. As regards the latter, to which attention has specially been called by Sullivan,⁹ there is certainly much more of it than would appear from the foregoing notices, for

¹ 'Neues Journ. der ausländ. med.-chir. Litteratur,' ix, Heft 1.

² 'Krankheiten des Orients,' 325.

³ 'Arch. de méd. nav.,' 1868, Févr., 82.

⁴ 'Dubl. Quart. Journ. of Med. Sc.,' 1871, Aug., p. 101.

⁵ 'Darstellung der Krankheiten in den Tropenländern,' &c., Würz., 1855, 186.

⁶ 'Arch. de méd. nav.,' 1882, Avril, 272.

⁷ 'Glasgow Med. Journ.,' 1877, July, 329.

⁸ 'Geogr.-med. Studien,' Berl., 1878, p. 172.

⁹ 'Med. Times and Gaz.,' 1875, Aug., p. 233.

the reason that I have disregarded all those accounts from the tropics which relate to the enormous prevalence of anæmia in general without mentioning chlorosis in particular, although there is no doubt that in many of them it is chlorosis that is particularly meant.

Goldschmidt's¹ opinion is that in Oldenburg the malady occurs mostly on a sandy soil, while in the marshes it is so seldom seen that an experienced practitioner from the marsh-country assured him that he had had no cases of chlorosis among his patients; but I am unable to decide from the recorded facts whether that experience expresses the general case, or how far a *swampy soil* gives immunity from the disease. In Norway, according to Axel Lund's observation, the malady is much rarer in the mountain valleys than on the *level country*; but the explanation of that, he thinks, is not to be found in the soil, but in the manner of life of the inhabitants. So far as I can judge from authentic observations, it is not at all probable that *racial or national peculiarities* create either a predisposition to chlorosis or an immunity from it.

§ 181. MOSTLY A MALADY OF WOMEN LEADING AN INDOOR LIFE.

'The *morbid diathesis* from which chlorosis develops, is undoubtedly a congenital one in many cases, sometimes associated with imperfect development of the vascular system (Virchow⁷), sometimes perhaps traceable to a morbid habit of body of the parents due to drinking, consumption, syphilis, and the like (Lund). But there are other cases in which the diathesis is acquired, having its basis in the individual's manner of life. There is scarcely a single fault in the kind of life that men, and more particularly women, lead both socially and intellectually, that has not been regarded, either by itself or in company with other things, as the cause of the acquired chlorotic diathesis. That the *social position* of the individual has, generally speaking, only an indirect in-

¹ 'Häser's Arch. für die ges. Med.,' 1845, vii, 315.

² 'Ueber die Chlorose,' &c., Berlin, 1872.

fluence at the most, may be inferred from the fact that chlorosis is distributed somewhat uniformly among the poor and the rich, among persons in all sorts of occupations, and among the inhabitants of town and country. "It is not on increasing poverty," says Huss, "that the general diffusion of chlorosis in Sweden depends; for, although there too the number of the poor has increased, that applies more to towns than to the country districts; while it is in the latter that the malady has appeared as something new, and moreover has shown itself more often among the moneyed classes than among the poorer class and the labourers."

The preponderance of chlorosis in the *female sex* indicates that there must be some factor predisposing to that type of disease, which is inherent in the female organisation; and that there are certain harmful things in their manner of life which are calculated, where such predisposition exists, to bring out those irregularities of blood-making which are the fundamental thing in chlorosis. Many observers are agreed that there is hardly any of these harmful influences which plays so great a part as prolonged confinement indoors, or *want of exercise in the open air*. With reference to the disease in Sweden, Huss says: "One of the principal causes is to be found in the completely changed mode of living which has taken place among the women part of the population in north country districts. Formerly the wife shared with the husband almost all the labours of the field; she lived, accordingly, almost constantly in the open air, undertook the most fatiguing kinds of work, and so became hardy and strong of body. But within the last twenty or thirty years, things have changed very much. The women now occupy themselves, except in harvest time, with household work almost exclusively; and their children also they keep for the most part shut up in their houses, where the cramped, narrow and unwholesome rooms must of themselves exert an unfavorable influence on the wellbeing of the inmates. But the great importance of these circumstances for the endemic existence of chlorosis is shown by the fact that the malady is rare or altogether unknown in those regions, such as Dalarne, Wermland, Dahlsland, and Småland, where the old customs continue to the present day; while, on

the other hand, it is most widely spread in the provinces, such as Westergothland and Halland, where the women are occupied almost entirely with household work." In like manner Lund, for Norway, would find the most potent although not the only cause of the ever-increasing chlorosis there, in the want of open-air exercise; and he points out that the difference in the amount of the sickness in the two sexes is partly explained by the fact that the boys, after their first childhood is over, are more in the open air than the girls. The real cause of the chlorosis which is endemic in the island of Miquelon, is stated by Gras to be "*la claustration presque absolue, à laquelle les jeunes filles se condamnent volontairement.*" Rigler found the disease in Turkey to be especially common among those Oriental women who led an inactive and purely sensual life in the harems. Pruner points out that chlorosis in Egypt occurs mostly in women who lead an indolent life behind latticed windows, such as the Jewesses in Cairo. In Algiers, also, according to the French physicians, it is very common among the Moorish women, who marry early and spend their lives in damp, closed-in houses, never leaving them except to walk in the gallery overlooking the dank and ill-smelling courtyard. Savarésy, speaking of the creole women in the West Indies, assigns their inactive and indolent mode of life, and the nervous excitability dependent thereon, as the causes of the chlorosis so common among them; and a similar conclusion is come to by Heinemann for Mexico and by Dudgeon for China.

In discussing the etiology of chlorosis, I must confine myself to a consideration of this one factor. It would carry me far beyond the limits that I have imposed on myself in this work, if I were to enter on a criticism of the many and various opinions and conjectures that have been put forward.

II. *Anæmia Intertropica.*

§ 182. THE GENERAL ANÆMIA OF THE TROPICS.

All observers agree that *anæmia* is a characteristic morbid phenomenon of tropical regions, and that as a symptom it is found mostly among the white residents, although it may be observed to no small extent among the coloured races also. As Sullivan says, anæmia constitutes "the foundation of nearly all diseases of tropical climates." There seems to be hardly any question that the peculiar influence of tropical climate which is called "relaxing," and which cannot be otherwise defined in physiological terms, is, for white people not habituated, a very material factor in the development of this disorder of nutrition. But it is clear that malarial influences contribute not inconsiderably to it, and also nostalgia, as Marchand¹ makes out for the convicts transported to Guiana from France. Experience shows, moreover, that the anæmic habit of body in white residents of the tropics communicates itself to their offspring; and therein lies the chief obstacle to the acclimatisation of the white race in the tropics.

Among the coloured races of the tropics "pure" anæmia is mostly the consequence of deficient food, or of a diet poor in animal substances, as well as of other debilitating influences such as the excessive use of spirits, bad dwellings, and the insufficient protection afforded by their clothing against the peculiarly trying tropical changes of weather. In other respects the accounts of the prevalence of anæmia among the coloured races of the tropics, particularly the negroes, are to be received with caution, for the reason that many of them relate, not to idiopathic poverty of blood, but to anæmia caused by *Anchylostoma duodenale* or the so-called "geophagia."

¹ 'Des causes et du traitement de l'anémie chez les transportés à la Guayane française,' Montp., 1869.

III. *Anæmia montana* (*Mal des Montagnes*).

§ 183. SYMPTOMS OF MOUNTAIN SICKNESS.

Under these and various colloquial names¹ there have been described a group of symptoms apt to come on during the ascent to more considerable elevations, or during any continuous residence there by persons accustomed to live at lower elevations or on the plains. They consist of a series of phenomena, proceeding partly from disorders of the respiratory and circulatory functions, and partly from an affection of the nervous system.

Usually the first thing noticed is a feeling of want of breath and of pressure on the chest, the respiration becoming hurried and wheezy, and there may even be a sensation of choking. It is clear that these phenomena are not to be attributed to the exertion of climbing the mountain, for they continue after the individual has arrived at perfect repose. Associated with them are a quickened pulse, palpitation, attacks of giddiness, pains in the head, sometimes even the feeling of going off in a swoon, disturbed or lethargic sleep, sickness or even vomiting, and usually a feeling of weakness or weariness which comes on in attempting to walk or otherwise exert the bodily strength, and which sometimes reaches so great a height that the slightest movement is torture. All these symptoms quickly abate whenever the descent is made to lower elevations; but if the individual remain at his elevated station they will persist, usually for a few days (two to four), but sometimes for several weeks and even months, until he has been thoroughly acclimatised. It is in the highest degree doubtful whether the seizure ever leads to serious or fatal symptoms, or, as Jourdanet alleges, to a chronic state of ill-health. Statements of that sort are probably based upon errors of diagnosis, or, in other words, upon wrongly attributing the observed morbid conditions to the effects of residence at a great height; at all events the severe forms of anæmia running a chronic course and with permanent after-effects, which some observers have attributed to mountain sickness, are due to quite other influences. MM. Gayraud and Domec, writing from Quito, say: "Nous avons vu arriver à Quito, et venant de niveaux inférieurs, des enfants, des vieillards, des femmes, des personnes de toute race, de toute complexion et de tout tempérament; jamais il ne nous a été donné de constater le moindre état morbide que l'on pu mettre sur le compte d'une acclimatation plus ou moins incomplète."

¹ In Peru and Quito these are: mal de la Puna, soroche, veta, mareo (*i.e.* mal de mer) de la Cordillera; in the Himalaya, bies or bootie.

It is only in cases of heart disease that the symptoms of mountain sickness appear to be of a rather serious kind. According to Guilbert's observations on the subject in Peru and Bolivia: "Quelques personnes atteints d'affection organique du cœur déjà avancée, ont souffert beaucoup plus longtemps et ont conservé pendant toute la durée de leur séjour la gêne de la respiration et de la circulation. Mais ce sont des faits exceptionnels."

Lastly, it is a noteworthy fact that the phenomena of mountain sickness have been observed also in *beasts of burden* (horses, asses, and mules) which had come up from the plains to considerable elevations.

§ 184. LOCALITIES WHERE MOUNTAIN SICKNESS HAS OCCURRED.

The malady does not occur except at elevations more than 2000 metres (6500 feet) above the sea, and it does not manifest itself fully until a height of 3000 to 4000 metres (10,000 to 13,000 feet) is reached. In this way we account for the fact that the sickness has never been observed to last for any length of time except in tropical countries, as it is only in these that a somewhat prolonged or continuous stay at so great elevations is possible. Most of the observations on mountain sickness, accordingly, and these the most thorough, come to us from the Cordilleras of the Western Hemisphere: from the *Rocky Mountains*,¹ the *Table-land of Mexico*² (*Anahuac*), certain elevated points of *Central America*, *Ecuador*³ (*Quito*), *Bolivia*,⁴ and *Peru*.⁵ In

¹ Fremont, 'Narrative of the Exploring Expedition to the Rocky Mountains,' Lond., 1846.

² Glennie, 'Philos. Magazine,' 1828, June, 149; Wuillot, 'Presse méd. belge,' 1866, Nr. 40; Jourdanet, 'Le Mexique et l'Amérique tropicale, &c.,' Par., 1864, 221; Coindet, 'Mém. de méd. milit.,' 1866, Mai, 423.

³ Bouguer, 'Mém. de l'Acad. des sc.,' Année 1744, Par., 1748, 261; de la Condamine, 'Voyage à l'Equateur,' &c., Par., 1751, 34; Remy, 'Annal. de voyages,' Par., 1857, clviii, 320; Gayraud et Domec, 'Montpellier médical,' 1878, Juin, 491.

⁴ Wadell, 'Voyage dans le Nord de la Bolivie, &c.,' Par., 1853; Burmeister, 'Reise durch die Plata-Staaten, u. s. w.,' Berl., 1861, ii, 263; Guilbert, 'De la phthisie pulmonaire . . . au Pérou et en Bolivie,' Par., 1862, 23.

⁵ Jose d'Acosta, 'Histor. natural y moral de las Indias.' French translation. Par., 1600, 90 (the first description of the malady, in which we find it already spoken of as "mal des montagnes"); Ulloa, 'Physik. und histor. Nachrichten vom südl. Amerika,' i, 73, 256; Cunningham, 'Lond. Med. Gaz.,' 1834, May, August; Pöppig, 'Reise in Chile, Peru, u. s. w.,' Leipzg., 1836, 84; Smith, 'Edinb. Med. and Surg. Journ.,' 1842, April, 357; Tschudi, 'Oest. med. Wochenschr.,' 1846, 601; 'Wien. med. Wochenschr.,' 1859, Nr. 6.

the Eastern Hemisphere the malady has been often observed in ascents of the *High Alps*,¹ and of *Arrarat*² (and *Elburz*) ; still more often in ascents of the *Himalayas*,³ and on the *Neilgherries*⁴ at elevations of no more than 2500 metres (8000 feet).

§ 185. THEORIES OF THE CAUSE OF MOUNTAIN SICKNESS.

Regarding the *cause of mountain sickness* there was much obscurity for a long time. Some thought that it was a kind of narcotic effect produced by metallic or vegetable poisons. Others sought for the reason of the phenomena in circulatory disorders induced by the rarefied air in persons not habituated to living in it ; as late a writer as Guilbert expressed the opinion that the tension of the free gases of the blood was increased under a lower weight of atmosphere, whereby pressure was exercised on the walls of the vessels. The most recent and generally received view is that the physiological disturbances are a consequence of changes in the composition of the blood due to the diminished quantity of oxygen which it receives, corresponding to the rarefied state of the air,—“*diminution de l'oxygène dans le sang par défaut de pression*,” as Jourdanet says in explaining the name “*anoxyhémie*” which he has given to the disease,—and that the disorder of the functions is gradually overcome by the respiration becoming deeper and more frequent. A modification of this theory of what takes place in mountain sickness has been adopted by Paul Bert.⁵ He has satisfied himself, by experiments, that the co-efficient of absorption

¹ De Saussure, ‘*Voyage dans les Alpes* ; Forbes, ‘*Travels to the Alps of Savoy*,’ Edinb., 1843, 223, and many other travellers ; see Meyer-Ahrens, ‘*Die Bergkrankheit*, u. s. w.,’ Leipzig., 1854, 40 ff.

² Parrot, ‘*Reise zum Arrarat*,’ Berl., 1834, i, 133 ff.

³ Moorcroft, ‘*Asiat. Researches*,’ Lond., 1818, xii, 413 ; Fraser, ‘*Journey through part of the Snowy Range of the Himalaya Mountains*,’ Lond., 1820 ; Jacquemont, ‘*Voyage dans l’Inde*,’ Par., 1841, ii, 260 ; Hoffmeister, ‘*Briefe aus Indien*,’ Braunsch., 1847, 242 ; Drew, ‘*The Jummo and Kashmir Territories*,’ Lond., 1875.

⁴ Collins, ‘*Ind. Annals of Med. Sc.*,’ 1860, Nov., 7 ; Mackay, ‘*Madras Quart. Journ. of Med. Sc.*,’ 1861, July, 29.

⁵ ‘*Compt. rend.*,’ 1882, tom. 94, No. 12, p. 805.

of hæmoglobin for oxygen does not exceed 10 to 12 per cent. in animals living at the sea level, whereas in animals domesticated to great elevations or under lower atmospheric pressure—the animal's blood examined by him came from La Paz, in Bolivia, at a height of 3700 metres or 12000 feet—it rises to between 17 and 21 per cent. Probably the case is the same with men; and, in Bert's opinion, the acclimatisation of men and animals removed from the plains to great elevations depends upon an increasing capacity of the hæmoglobin for absorbing oxygen, so that disorders caused by the diminished quantity of oxygen in the air are gradually overcome.

Another point, specially emphasised by Gayraud and Domec, is that the malady known by the name of mountain sickness is slight or severe according to the *idiosyncrasy* of the individual. In their own persons they observed no symptoms of it on coming to Quito; and in the case of some other strangers arriving in Quito they have either failed to observe it altogether or have seen only very slight degrees of it.¹

¹ See also the earlier observations on the same subject by Meyer-Ahrens, l. c., p. 128.

CHAPTER XV.

SCURVY.

§ 186. NEGLECT OF HISTORICAL RESEARCH ON SCURVY.

“It is a remarkable fact that, with all the zeal which historical students of medicine have applied to the more important and more widely distributed forms of national sickness, there has not been in recent times a single thorough inquiry into the *history of scurvy*. It has in most cases been thought sufficient hitherto to engage in unprofitable discussions as to whether scurvy was known to the ancient and mediæval practitioners of medicine; whatever the medical writers of the sixteenth and seventeenth centuries had alleged of the disease down to their own time has been allowed to pass current without scrutiny as sterling coin; and from those materials a representation has been produced of the natural history of survy, which has been copied and re-copied from the one or two monographs on the subject into nearly all the later compendiums of medicine. In the whole of the more recent literature I know of only one author who has taken the trouble to inquire into the history at first hand and to subject an obscure heap of materials to the light of criticism. I refer to Lind, whose admirable treatise¹ still holds a foremost place among the writings on scurvy; although all the later authorities, with the exception of Sprengel,² have paid no attention to the important indications concerning the history of scurvy which the book contains. It seemed all the more necessary, therefore, that I should make this

¹ ‘A Treatise on the Scurvy,’ Edin., 1752. An alphabetical list of authorities quoted in the text is given at the end of the chapter.

² ‘Geschichte der Arzneikunde,’ iii, 93.

matter the subject of a new and thorough scrutiny, and should exhibit it in a somewhat broader light; and this was not the less incumbent on me because the results of my inquiry proved to be in many respects a good deal at variance with the view of the facts hitherto accepted."

These are the words with which I began the chapter on scurvy in the first edition of this work, by way of justifying the detailed treatment of the subject from its historical side. Since that was written several considerable works on scurvy have appeared, in which the history of the malady has received attention; in these I find the results of my researches reproduced, and no material exception taken or contradiction given to them. I think myself warranted, therefore, in using them as the basis of the historical survey of the disease in this second edition, all the more so that the facts which have come to my knowledge in the interval have only served to confirm me in my original opinions.

§ 187. CRITICISM OF THE SUPPOSED REFERENCES TO SCURVY IN ANCIENT WRITINGS.

The writings that have come down to us from antiquity and the middle ages give no help towards deciding whether scurvy occurred, or was known to medicine, in those times at all; or, if so, to what extent. In order to make out that the Graeco-Roman and Arabian physicians were acquainted with scurvy, special emphasis has been laid on a form of disease described under the name of "*lienes magni*" by Hippocrates,¹ Celsus,² Aretæus,³ Cælius Aurelianus,⁴ Paulus Ægineta,⁵ Avicenna⁶ and others.

"*Οκόσοι δὲ σπληῖνα ἔχουσι μέγαν,*" says the Hippocratic writer in one of the treatises, "*όσοι μὲν εἰσι χολώδεις, κακοχροοί τε γέγονται καὶ κακελκείες καὶ δυσώδεις ἐκ τοῦ στόματος*

¹ See the passages quoted in the text.

² Lib. ii, cap. vii, ed. Targa, i, 54.

³ '*De causis morb.*,' lib. i, cap. xiv, ed. Kühn, 110.

⁴ '*Morb. chron.*,' lib. iii, cap. iv, ed. Amman, Amstelod., 1755, 448.

⁵ Lib. iii, cap. xlix, ed. Lugd., 1551, 222.

⁶ '*Canon*,' lib. iii, Fen. xv, Tract. i, cap. iv, ed. Venet., 1564, i, 790.

⁷ '*De affectionibus*,' § 20, ed. Littré, vi, 228.

καὶ λεπτοί. καὶ ὁ σπλὴν σκληρὸς, καὶ αἰὲ παραπλησίως τὸ μέγεθος. καὶ τὰ σιτία οὐ διαχωρεῖ.” And in another passage,¹ “οὐλα δὲ πονηρὰ καὶ στόματα δυσώδεα οἷσι σπλῆνες μεγάλοι. Ὀκόσοι δὲ ἔχουσι σπλῆνας μεγάλους, μήτε αἰμορῥαγίαι γίνονται μήτε στόμα δυσώδες, τουτέων αἱ κνῆμαι ἔλκεα πονηρὰ ἴσχουσι καὶ οὐλὰς μελαίνας.

There is, in my view, but little reason to take that group of symptoms, characteristic of the “*lienes magni*,” as pointing to scurvy; firstly because swelling of the spleen, which was undoubtedly a constant occurrence in that malady according to the descriptions of observers,² is by no means one of the more commonly noted phenomena of scurvy; and secondly because the Hippocratic writers themselves³ refer the origin of the malady in question to its true source—the malarial cachexia. This view of mine is further supported by the fact, to be afterwards adduced, that scurvy and malarial cachexia have often been confounded in later times.

Other authorities⁴ have detected scurvy in the form of a disease mentioned by several writers of antiquity under the name of “*stomakake*” and “*skelotyrbe*,” supporting their contention by a reference to Pliny’s⁵ account of a disease which was prevalent in the Low Countries among the Roman army under Germanicus, and to Strabo’s⁶ narrative of an outbreak in the Roman army under Ælius Gallus in Arabia.

“In Germania trans Rhenum castris a Germanico Caesare promotis,” says Pliny, “maritimo tractu fons erat aquae dulcis solus, qua pota intra biennium dentes deciderent, compagesque in genibus solverentur. Stomacacen medici vocabant et sclerotyrben ea mala. Reperta auxilio est herba, quae vocatur Britannica, non nervis modo et oris malis salutaris, sed contra anginas quoque et contra serpentes. . . Frisii qua castra erant, nostris demonstravere illam; mirorque nominis

¹ ‘*Praediction.*,’ lib. ii, § 36, ed. Littré, ix, 66.

² See the excellent description in Aretaeus.

³ For example, in the genuine Hippocratic treatise, ‘*De aere, aquis et locis*,’ § 7, ed. Littré, ii, 26, where it is stated that the disease of *σπλῆνες μεγάλοι* is most frequent in damp and marshy situations, and that dropsy usually supervenes on the general wasting. See also ‘*Lib. de affect. intern.*,’ §§ 30-34, ed. Littré, vii, 245, *seq.*

⁴ Such as Guyon in still recent times (‘*Compt. rend.*,’ 1846, Juin, 29; ‘*Gaz. méd. de Paris*,’ 1846, No. 27, p. 533.

⁵ ‘*Hist. nat.*,’ lib. xxv, cap. vi, ed. Franz, Lips., 1788, vii, 641.

⁶ ‘*Geogr.*,’ lib. xvi, towards the end of the book.

causam, nisi forte confines Oceano Britanniae, velut propinquae, dicavere."

Some have gone still farther, and have deduced from this recommendation of the herba Britannica as a remedy for "stomakake" and "skelotyrbe" the conclusion that a diseased state of the mouth, mentioned by Marcellus¹ under the name of "oscedo," was also scurvy, the remark of Marcellus being, "oscedinem herba Britanica viridis sumpta in cibo, lactucae modo, sanat."

Of the nature of this "stomakake," there is nothing to be learned except from Pliny's account, which has a somewhat romantic ring. It is not impossible that this affection of the mouth was a symptom of scurvy; but it is just as probable that the disease in the Roman army was the same that has been seen in modern times and described under the name of "stomatite ulcéreuse," as an endemic or epidemic malady among the troops of more than one European power, and particularly often among the French troops.

As to the nature of the "skelotyrbe" and its connexion with the affection of the mouth, I am unable to form any opinion.

Galen² gives the following definition of it:—"Skelotyrbe species est paralyseos, qua quis recte ambulare non potest et latus alias in rectum, quandoque sinistrum in dextrum, aut dextrum in sinistrum circumfert, interdum quoque pedem non attollit, sed attrahit velut iis, qui magnum quid adscendunt."

In this description we are reminded somewhat of the gait of tabetic patients. So far as relates to the recommendation of herba Britannica, nothing can be inferred either regarding "stomakake," or regarding "oscedo" (which is mentioned by no other author), for the reason that it is not known what plant is meant. Euricius Cordus³ conjectured that it is Bistorta which is intended, but he afterwards gave up that opinion. Agricola⁴ does not commit himself to an opinion of his own, and merely states the view of Cordus.

The idea, started in modern times by Seidlitz,⁵ and adopted by Hecker, that the disease described by the physicians of antiquity under the name of "morbus cardiacus

¹ 'De medicamentis,' cap. xi, in Stephani, 'Collect.,' 291.

² 'Definit. med.,' § 293, ed. Kühn, xix, 427.

³ 'Botanologicon,' Colon., 1534, 137.

⁴ 'Med. Herbariae,' lib. ii, Basil., 1539, 56.

⁵ In Hecker's 'Wissenschaftl. Annalen der Heilkde.,' 1835, xxxii, 129.

s. coeliacus" corresponded to pericarditis scorbutica (found mostly in Russian practice), has been completely overthrown by Landsberg.¹

After a close scrutiny of the ancient and mediæval medical writings, I have found only one description of a form of disease which so far corresponds to the picture of scurvy, that the identity of the two may be hazarded. I refer to the malady spoken of in the Hippocratic collection² under the name of ἐλκὸς αἱματίτης; of which it is said:

“Τάδε δὲ ἐν τῷ νοσήματι προσγίνεται· ἐκ τοῦ στόματος κακὸν ὄζει, καὶ ἀπὸ τῶν ὀδόντων τὰ οὖλα ἀφίσταται, καὶ ἀπὸ τῶν ῥινῶν αἷμα ῥέει. Εἰρίοτε δὲ καὶ ἐκ τῶν σκελέων ἔλκεα ἐκφλυνδάνει, καὶ τὰ μὲν ὑγιαίνεται, τὰ δὲ ἄλλα προσγίνεται, καὶ ἡ χροὴ μέλαινα, καὶ λεπτόδεσμος· περιφοιτῇ δὲ καὶ τάλαιπωρέειν οὐ πρόθυμος.”

It is *a priori* highly probable that scurvy had been epidemic from time to time in antiquity under the same circumstances that have given rise to it in the modern period or in recent times. It certainly follows from the account given by Jacques de Vitry³ of a disease called by him the plague, which ravaged the army of the crusaders before Damietta in 1218, and from Joinville's⁴ description

¹ 'Janus,' 1847, ii, 53.

² 'De affect. intern.,' § 46, ed. Littré, vii, 280.

³ Liv. iii, § 351, 'Collect,' Guizot (quoted by Marchand, 'Étude histor. et nosol. sur quelques épidémies et endémies du moyen âge,' Par., 1873, 17): "Un grande nombre d'hommes de notre armée furent en outre saisis d'une certaine peste contre laquelle les médecins ne pouvaient trouver aucun remède dans leur art. Une douleur soudaine s'emparait des pieds et des jambes: aussitôt après les gencives et les dents étaient attaquées d'une sorte de gangrène, et le malade ne pouvait plus manger. Puis l'os de la jambe devenait horriblement noir et ainsi après avoir souffert de longues douleurs pendant lesquelles ils déployèrent une grande patience, un grand nombre de chrétiens allèrent se reposer dans le sein du Seigneur. Quelques-uns étant parvenus à gagner le printemps se guérirent alors par l'effet des chaleurs."

⁴ 'Histoire de Saint-Loys,' Par., 1617, 121. "Nous vint une grant persécution et maladie en l'ost; qui estoit telle que la chair des jambes nous desséchait jusqu'à l'os, et le cuir nous devenoit tanné de noir et de terre à la ressemblance d'une vieille houze, qui a été longtemp mucée derrière les coffres. En oultre, à nous autres qui auions cette maladie, nous venoit une autre persécution de maladie en la bouche, de ce que nous auions mengié de ces poissons, et nous pourrissoit la chair d'entre les gencives, dont chacun estoit orriblement puant de la bouche. Et à la fin guesres n'en enchappoient que tous ne mourussent. Et le signe de mort que l'on y congnoissoit continuellement estoit quand en se prenoit à saigner du neys, et tantoust on estoit bien asseuré d'estre mort de brief."

of the sickness that broke out in 1250 among the army of Louis IX at the siege of Cairo, that scurvy had existed long before we have any medical recognition or description of it as a peculiar form of disease.

§ 188. HISTORICAL EPIDEMICS. THE "SCORBUTIC CONSTITUTION" OF FORMER TIMES A MYTH.

The history of scurvy as an epidemic malady well known to the medical profession does not begin before the fifteenth century, or the period of the Renaissance—a movement which touched every relation of life, and by exciting an interest in foreign countries, gave occasion to sea voyages on a scale never before known.

As early as the middle of the fifteenth century we find in the history of maritime commerce accounts of expeditions to remote regions which had only a partial success or even proved total failures owing to scurvy breaking out among the crews. One of these was the great expedition of Vasco de Gama, on board whose ships the disease appeared off the African coast in January, 1498, in so malignant a form that he lost fifty-five of his fellow-adventurers in a short time.¹ Other examples are Cartier's unfortunate expedition² in 1535, the expeditions to Canada under v. Monts, Pontgrave and Poutrincourt³ towards the end of the sixteenth century, the French naval expedition to India under Dellon,⁴ Admiral Anson's voyage round the world with an English fleet from 1740 to 1744 (during which the disease broke out at various times in different latitudes⁵), the voyage of Ellis⁶ in 1746-47 to Hudson's Bay to discover the North-West Passage, the expedition of the English fleet to the coast of Algiers⁷ in 1773, the cruise of the Channel Fleet in 1780⁸ under Admiral Geary,

¹ Ramusio, 'Raccolta delle navigaz. e. viaggi,' i, 119.

² Hakluyt, 'Principal Navigations, &c.,' Lond., 1598, iii, 225.

³ 'Collection of Voyages,' iii, 808.

⁴ 'Voyage aux Indes orient.,' quoted by Lind, 557.

⁵ Walter and Robins, 'Voyage round the World,' &c., Lond., 1748.

⁶ Ellis, 'Voyage to the Hudson's Bay,' &c., Lond., 1748.

⁷ Aaskow, 'Diarium med. navale,' Lond., 1774.

⁸ Armstrong, l. c., 4.

when he was obliged to put back to England with two thousand four hundred men down with scurvy, and the voyage of a convoy of English ships round the Cape of Good Hope in 1781.¹

It was not until the end of last century that attempts were made by the naval powers chiefly concerned, particularly England, to carry out such rules in the fitting out of ships for great naval enterprises, as experience had shown to be best adapted to prevent the outbreak of the malady; and, as a matter of fact, scurvy on board ship, and especially in ships of war, has been seen during the present century much more rarely than in former times, although there have not been wanting epidemics at sea even in the most recent times, under circumstances to be afterwards mentioned.

Of somewhat later date are the first authentic accounts of the epidemic occurrence of scurvy on land. The earliest mention of the nosological term "Scharbock" is to be found in Cordus,² who, in speaking of the healing virtues of the *Chelidonium majus*, refers to its efficacy in scurvy, the herb being known among the "Saxones" (Low Saxons or inhabitants of the North German plain)³ as "Scharbocks-Kraut;" but it does not appear that he had ever seen the disease itself. Shortly after there appeared the statements of Olaus Magnus,⁴ concerning the often observed epidemic prevalence of scurvy in the Scandinavian kingdoms, especially in times of famine; and about the same time, or a little later, the writings of Echthius, Ronsseus, Wier, Dodonaeus and Brucaeus, which testify to the comparatively common occurrence of the disease along the littoral of the North Sea and the Baltic (as Brucaeus says: "*Morbus maris Baltici, Finnici et Bothnici sinus accolis, iisque qui Germanico Oceano adjacent, Saxonibus, Phrygiis, Batavis, totique Scaniae sive Scandinaviae, quae Danos, Norwegos, Suecos complectitur, familiaris*").

These excellent descriptions of the malady, more especially by Echthius, Ronsseus and Wier, leave no doubt as to its nature. Also in the epidemics of 1556 and 1562 in the

¹ Curtis, l. c., 9.

² L. c., p. 94.

³ It is clear from the treatise of Brucaeus, quoted in the text below, that it is the inhabitants of that tract of country who are meant.

⁴ 'De gentium septentrional. conditionibus,' &c., Romæ, 1555, lib. xvi, cap. 51.

Netherlands, which are mentioned by Dodonaeus, the disease was certainly scurvy and not ergotism, as some have been inclined to think from a remark of the chronicler as to the injurious effects of damaged grain which had been imported from Prussia. On the other hand, it remains a question whether the disease at that time was an important one among the national maladies of those countries; the fact that so experienced and widely consulted a physician as Foreest¹ betrays only a slight acquaintance with scurvy, warrants us in concluding that the epidemic outbreaks of it, at least, had reached no very great extent.

These observations had hardly become known to the medical profession at large before the dogmatism of the schools laid hands upon them. A succession of medical treatises came out, in every page of which one may discover that their authors had in all probability never had an opportunity of seeing a single case of scurvy; and thus there was initiated in the course of a few years one of the most foolish episodes in the whole history of medical science or practice. "Scurvy" became the Alpha and Omega of professional routine, the catchword of the day, "the asylum ignorantiae of the practical man," as Baldinger excellently puts it. And although a few of the more sensible observers, like Willis, Sydenham, Hoffmann and Kramer strove against that misuse of the word and idea, yet it persisted long into the eighteenth century; until at length an impartial scrutiny and correct estimation of the facts brought the empire of scurvy within narrower and narrower limits. Then it happened, as it has often happened in similar revolutionary movements in the subject-matter of our science, that scepticism fell into the opposite extreme. The reality of facts well authenticated began to be doubted altogether, and it was a question whether scurvy should not be struck out absolutely from the list of specific forms of disease. Into this chaos the first beams of light fell when Lind's classical work appeared. In the end scurvy was secured its rightful place in the nosology through the extremely careful way in which the observations on it had been made, especially in the Baltic Pro-

¹ 'Observ. et curat. med.' lib. xx, obs. xi, Lugd. Batav., 1595, p. 347. In this passage he even speaks of scurvy as "morbus rarus."

vinces of Russia. But that old-world phantom of a Scorbatic Constitution still continues to haunt the brains and books of a good many practitioners. For the reason that the disease has occurred comparatively seldom in recent times, they discover that scurvy is declining or gradually dying out, not being aware that the decline is for the most part only an apparent one, brought about in the way already adverted to. A few references to the literature of the subject in the seventeenth and eighteenth centuries will suffice to prove that the view here taken is a correct one, although it is in almost direct opposition to opinions hitherto current.

The earliest information of the prevalence of scurvy in other countries besides the coast regions above mentioned dates from the year 1486, when "Scharbock," according to several chroniclers, would appear to have shown itself in some parts of Saxony, Thuringia, and the adjacent country as a malady previously quite unknown there. It is not difficult to follow up this assertion to its source if we compare among themselves the various chronicles which record the fact. We find the first indications of it in the 'Annales urbis Misnicæ' of Fabricius, who died at Meissen in 1571 in the office of rector of the Fürstenschule; the passage runs: "Grassatus est hoc anno novus et inauditus in his terris morbus, quem nautæ Saxonie vocant den Scharbock, qui est inflammatio in membris partium carnosarum, cui quoque celerius adhibetur medicina, eo citius malum restinguitur. Sin mora accedit paullo tardior, sequitur membri affecti mortificatio, quam siderationem nostri, Græci σφάκελον dicunt, ultimum gangraenæ malum. Nam caro ab ossibus defluit et continua quoque a lue corrumpuntur."

In my view it is not so clear that the disease here is scurvy, and not rather ergotismus gangraenosus; for even in much later times we meet with the same confounding of the two diseases. We shall readily understand how Fabricius came to use that nomenclature when we reflect that he, like the members of the medical profession itself in his time, was still altogether unacquainted with the nature of ergotism; whereas the treatises on scurvy by Echthius and Ronssens¹ had already appeared, as well as Lange's historical researches.² Fabricius, therefore, was ready to identify by its name the pestilence of 1486 with the disease which had lately come to knowledge, all the more so that a remote resemblance between certain of the phenomena of disease will satisfy the lay mind even in a question of fact.

In 1589 Brunner² published a tractate on the scurvy which does little more than reproduce the statements of Wier. Whether he had ever

¹ Antwerp, 1564.

² Basel, 1554.

³ 'De Scorbuto,' tract. ii, appended to Brucaeus, ed. cit.

seen scurvy does not appear from anything in his work; still less is there anything in it to justify the inference that scurvy had been endemic or epidemic in his own country of Saxony. A few years after came the work of Albertus¹ who, in giving the distribution of scurvy, enumerates the coast lands of the German Ocean and Baltic Sea, as stated by Brucaeus and others, and then adds that the malady had begun to spread into the adjoining inland territories, "quibus hactenus insolens fuit" (wherein he contradicts the assertion of Fabricius), and that it had shown itself in particular in Silesia, Bohemia, and Saxony. Whether he had himself ever seen cases of scurvy his book does not make clear. If neither of the works already mentioned may be said to possess any original value, they are none the less good compilations. But in the next book that came out, the treatise by Eugalenus,² we are introduced to a piece of make-believe which is, in two respects, without a rival in the whole literature of medicine: firstly, in the ignorance of its author, and secondly, in the results which the book achieved notwithstanding. For more than a century it continued to be the canonical book for the doctrine of scurvy, even the best physicians of the time being unable to keep themselves free from its tyrannical influence. There can be only one explanation of this fact—that the disease was on the whole rare, occurring only within small circles, and coming under the notice of those practitioners least who wrote about it most. Eugalenus took from the writings of his predecessors the name of the disease, and from Wier he took a brief epitome of the clinical characters; but beyond that he developed his notion of scurvy in the most arbitrary manner out of his own head, and applied it so generally to diseases that in the end the whole nosology was received therein. This theory, founded on the crassest of dogmatism, he built up with an arbitrariness of assertion and assumption beside which the fabrication of the Galenic doctrine of the Qualities is mere child's-play. The real phenomena of the disease—the affection of the gums, the ecchymoses, hæmorrhages and the like—vanish for diagnostic purposes before the truly pathognomonic symptoms which he discovered in certain characters of the urine and of the pulse; by these he recognised the disease, altogether irrespective of the presence or absence of those casual accompaniments, which are essentially the scorbutic symptoms. One can understand how a production of that sort might exert much influence at a time when science had not yet shaken off its stiff chains of dogmatism, and when the opportunities of observing the disease itself, few at any rate, were for that reason but little turned to account in testing the theory. Thus it is that we actually find the men who then led the tone in the German medical world, such as Horst³ and Sennert,⁴

¹ 'Scorbuti historia,' Wittbg., 1594. Printed in Sennert, Tract. 354.

² 'De morbo scorbutico liber,' Hagae-Com., 1658.

³ 'Observ. med.,' lib. vii, 34; Opp. Norimb., 1660, ii, 364.

⁴ 'Tract. de Scorbutico,' Wittbg., 1624; also in 'Pract. Med.,' lib. iii, part v, sect. ii, Wittbg., 1648, 542.

coming forward as faithful henchmen of Eugalenus: "*Tanta omnino morborum et symptomatum farrago in hoc affectu concurrir,*" cries Sennert, "*ut vix alius sit tam πολύμορφος et qui sub tot morborum speciebus latitet, ac Medicos, etiam cum cavisse maxime videntur, saepe decipiat et deludat.*"

Shortly after comes Drawitz¹ with a doleful book, in the preface to which he declares that all mankind will soon be scorbutic, for most children are conceived in scurvy and born with it. He breaks out violently upon those of the more sensible of his contemporaries who had pronounced "Scharbock" (as interpreted by himself and Eugalenus) to be a nonentity; while, among other paradoxes, he speaks of the scorbutic gout (p. 3) and of the scorbutic "Kriebelkrankheit" (p. 73). The next is Moellenbroeck,² who observes in the introduction to his treatise: "*Immo nullus fere jam morbus est, cui se non adjungat scorbutus, unde nisi antiscorbutica interdum reliquis admisceat medicamenta, vix eos curabit medicus.*"

After him comes Güldenklec,³ who certainly had opportunities of seeing scurvy somewhat frequently at Colberg, on the Baltic, where he resided; but who betrays in many passages⁴ so complete mystification as to what is implied in the notion of scurvy that one can place no reliance on his statement:⁵ "*Inter omnia, quibus corpus humanum expositum est, morborum κολαστήρια nullum scorbuto, oris hisce maritimis endemio, frequentius.*"

A later author, George Gottlieb Richter,⁶ is still found writing entirely in the Eugalenian sense: "*Scorbutus non tam morbus est, quam morborum illas, certe ob symptomatum copiam et versatilem indolem tam multiplice facie apparet, ut eam inter lineamenta, periti etiam manibus ductu, non nisi aegre agnoscas,*" and elsewhere as well he follows Eugalenus closely. C. J. Lange⁷ makes the same profession of faith: "*Faciem hujus affectus quod attinet, impossibile est illam accurate depingere; tam varias enim formas assumit, et nullus pene affectus detur in tota praxi, sub cujus pallio non quandoque personam suam agat.*"

The doctrine of scurvy fared hardly any better in the Netherlands, where the views of a Eugalenus or a Sennert found favour more readily than the unbiassed observations of an Echthius or a Wier. Barbette⁸ describes scurvy in terms which betray the influence of the German school. Beverovici⁹ does not hesitate to declare that in his time (first

¹ 'Unterricht vom Schmerz-machenden Scharbock,' Leipz., 1647.

² 'De varis seu arthritide vaga scorbutica tract.,' Lips., 1672.

³ 'Opp.,' Lips., 1715.

⁴ 'Epist.,' lib. iii, quaest. xx, ed. cit., 569, epist. xxiii, p. 585, &c.

⁵ 'Casuum medic.,' lib. iii, cas. 34, ed. cit., 143.

⁶ 'Diss. de Scorbuto,' Götting., 1744; in 'Opuscul. med.,' Frankf., 1780, i,

160.

⁷ 'Prax. med.,' cap. vii, § 4; 'Opp.,' Lips., 1704, ii, 38.

⁸ 'Prax. med.,' iv, cap. 3; 'Opp.,' Genev., 1688, ii, 153.

⁹ 'Opp.,' i, 91.

half of the seventeenth century) there were few persons free from scurvy. In the same way was the subject dealt with by Linden,¹ and by Bontekœ,² who designates scurvy as “radicem et causam omnium morborum;” and even Boerhaave by no means attained to an unprejudiced view of scurvy, although he admits that the malady in his day (beginning of the eighteenth century) occurred more rarely in the Netherlands than would seem to have been the case formerly.

In Scandinavian countries the state of matters was little different, as we may learn from the report of the Academical Council of Health published in 1645; and I can only regard it as the result of superficial research that Ilmoni, speaking in his “Nordens Sjukdoms-Historia” of the state of health in the sixteenth and seventeenth centuries should say: “This much may safely be alleged, that scurvy during those two centuries was the predominant chronic national malady of the northern countries, the fundamental dyscrasia which characterised that period in Scandinavian lands, and imparted to every form of sickness there a uniform specific background of its own colour.” As evidence of the extent to which men could carry the idea of scurvy even in much later times, I quote the following fact relating to the year 1808 from Arnold, who was at that time a surgeon in the English fleet which had joined the Swedish fleet for a cruise in the Baltic. On information being sent that scurvy was raging in a disastrous form on board the Swedish men-of-war, Arnold was transferred to them in order to accompany them home. He soon satisfied himself, however, that there was not a single trace of scurvy, but that the crews were suffering from well-marked typhus.

Finally in England the doctrine of scurvy fared little better. We find Lister, for example, holding strictly to the point of view of Eugalenus, whose services to the State and to science he could not extol highly enough; and even in Bisset, who wrote about the middle of the eighteenth century, we read: “The diseases induced by more or less of the scorbutic cacochymy in these different circumstances are chiefly the following: viz. an habitual land-scurvy or scorbutic affection of the first class; slow scorbutic or nervous little fevers of long duration which are often attended with hypochondriac and hysteric symptoms; scorbutic or erysipelatous defluxions; scorbutic rheumatisms; scorbutic eruptions of various sorts; the gout; the sciatica; palsies; hypochondriac and hysteric affections; cachexy and dropsy; or an atrophy.”

The medicine of France and Italy from the sixteenth to the eighteenth century concerned itself with scurvy on the whole very little; the slight-

¹ ‘Medulla Med. Pars. pathol.,’ Franecker., 1642, 35, 112.

² ‘Opp.,’ Amsterd., 1689, ii, 138.

³ In Bartholini, ‘Cist. med.,’ Hafn, 494.

⁴ ‘Bidrag till Nordens Sjukdoms-Historia,’ Helsingfors, 1853, iii, 4.

⁵ ‘Lond. Med. and Phys. Journ.,’ 1809, xxi, p. 17.

⁶ ‘Tract. de morb. chron.,’ 1696, p. 71.

⁷ ‘Medical Essays and Observations,’ Newcastle, 1766, p. 197.

ness of the knowledge of it in those countries may be inferred from the pathological inquiries of Fracassini,¹ who designates scurvy as a variety of hypochondria, a view that was taken of many things by the solidist school of pathology in those times.

There were bound to be, as we have said, a few sensible and observant men who saw through the abuses that were being practised in this manner with names and notions. A reaction at length set in, which went so far as to deny altogether that scurvy existed as a specific form of disease. That much we may infer from the replies to their opponents by those implicated in the scurvy mystification. The reactionary party is but scantily represented in the literature of scurvy; although there are among them men whose word should have weighed heavily in the scale, and whose testimony remains of special importance for the purposes of the historian. Sydenham² says: "*Licet non dubitem, quin Scorbutus in his plagis Borealiibus revera inveniatur, tamen eum morbum non tam frequentem, quam fert vulgi opinio, occurrere persuasum mihi habeo; multos autem ex iis affectibus, ne pluribus dicam, quorum nomine Scorbutum incusamus, vel morborum Fientium, nondum vere Factorum, quique nullum adhuc certum induerunt typum, effecta esse, vel etiam infelices reliquias morbi alicujus nondum penitus devicti, a quibus sanguis ceterique humores contaminantur . . . Et sane nisi hoc concedamus, Scorbuti nomen, uti hodie fit, in immensum crescet et omnem fere morborum numerum absolvet,*"—an apprehension which, as we have seen, the sequel fully warranted. Hoffmann's³ language is almost the same, and he adds a warning against the good being rejected with the bad and the existence of scurvy altogether denied. Mead⁴ says: "*Scorbuti nomen apud auctores medicos morbum designat tam multiplicem et facie diversum, ut non idem, sed alius atque alius esse videatur.*"

Kramer,⁵ who has a very good description of scurvy from cases which he himself saw, (although the disease, as he expressly tells us, was rare,) observes: "And accordingly it (scurvy) is so little known to these persons, particularly the acidists, that they turn the word '*scorbutus*,' to a monstrous use, and include therein all kinds of '*sordes caco-chymiae*,' even the lues venerea, not knowing how to discriminate one thing from another. In this way every Dutchman and Zeelander, every Dane and Swede, and in fact all the Northmen, are bound to be all alike scorbutic from their birth, or at least so constituted that in all their diseases scurvy is ever an ingredient."

So far, then, as relates to the data of sixteenth and seventeenth century writers concerning the prevalence of scurvy

¹ '*Opuscula pathol.*,' part iii, cap. ii, Lips., 1758, 368.

² '*Observ. med.*,' sect. vi, cap. v, '*Opp.*,' Genev., 1736, i, 172.

Med. ration. syst., tom. iv, part v, cap. i, § 1, '*Opp.*,' Genev., 1748, iii, 369.

⁴ '*Monita et praecepta med.*,' Lond., 1751, p. 123.

⁵ '*Medicina castrensis*,' Nürnberg, 1735, p. 77.

during that period, it seems to me to be quite obvious, according to the foregoing account of the matter, that we have small warrant to conclude from them that scurvy was universally diffused. But in judging of this matter, we have another and very notable circumstance, namely, that remarkably few records of epidemics of scurvy have come down to us. If we exclude all those data, in the chronicles or the epidemiological writings of the sixteenth and seventeenth centuries, which are obviously errors of diagnosis, such as confounding scurvy with ergotism¹ and malarial sickness,² there remains only a small number of epidemics of scurvy for that period, as the following table shows; and this is all the more noteworthy, that the period is far from poor in epidemiological records in general. Without taking this fact as absolutely conclusive, I think that it should at any rate confirm the impression derived from the foregoing criticism, that *scurvy in former centuries had by no means that importance as a universal malady which is assigned to it by contemporary and later writers*;³ although, for reasons to be afterwards given, it is probable that the malady was more common, perhaps also more widely distributed, in former times than during recent years.

¹ Errors of that sort occur in Sennert's book (p. 601), where he speaks of *gangraena scorbutica*; in Bonet's 'Sepulchretum' (ii, 338); and even in Hoffmann (l. c., § 8).

² In this second category are to be reckoned the notices by Sylvius (in the 'Tract. de affect. epid. anni, 1669,' § 471, 'Opp.,' Amstel., 1679, p. 842), by Fonesca ('Consult. med.,' Frankf., 1625, i, cons. ii, p. 31), and by Morley ('De morb. epid. observ.,' Lond., 1686). Morley describes, under the name of *febris epidemicus scorbuticus*, an epidemic of malarial fever in 1679, which was widely spread over England and the Netherlands. From this Ozanam (l. c., iv, 184) has made out an epidemic of scurvy; it is clear that he had read the book, but only in that uncritical temper which makes his own work of so little use. Others also have described it as such, faithfully following him. This laxity must appear all the more incomprehensible, inasmuch as we have another and excellent account of the same epidemic by Sydenham (Greenhill's ed., pp. 275, *seq.*).

³ Rütenbeck and Horn (l. c., p. 27), writing at the beginning of the seventeenth century, explicitly state that scurvy occurred very rarely as an epidemic in Germany, France, and other European countries, apart from war times and other circumstances leading to scarcity.

[*Chronological Table of Epidemics of Scurvy.*]

Time.		Place.	Authority.
1556 } 1562 }	...	Coast Provinces of the Netherlands	{ Wier, Ronsseus, Dodonaeus
1625	Summer	Breda (besieged fortress).....	v. d. Mye.
1631	...	In the Swedish Army before Nürnberg	Rötenbeck and Horn.
1632-33	Winter— Summer	Augsburg after its occupation by the Swedes	Höchstetter.
1699	"	Paris, in the Hôtel Dieu	Poupart.
1703	Summer	Thorn (besieged by the Swedes).....	Bachstrom.
1731	Spring— Summer	{ Cronstadt	Sinopeus.
1732	Summer		
"	Winter— Summer	Wiborg.....	Nitzsch.
1733	Spring— Autumn	Cronstadt	Sinopeus.
"	...	St. Petersburg, among troops from the Ukraine.....	Nitzsch.
1735	Winter— Spring	Temesvar, in the Imperial Army ...	Kramer.
1738	Winter— Summer	{ Voronej, among soldiers and sailors	Cork.
1739	"		
1740 } 1741 }	...	{ Stockholm, on board the men-of-war	{ Linnaeus.
1742	...	St. Petersburg.....	Buddeus.
"	...	Finland.....	Nitzsch.
1749	...	Riga, among the troops	Cork.
1750	Autumn	Corregliano, and other places in Venetia.....	Agostini.
" } 1751 }	Winter	Riga, among the troops	Cork.
1752	...	In a few localities near Verona	Targa.
1758	...	Breslau, after occupation by the Prussians.....	Baldinger.
1760 } 1761 }	...	{ Lower Silesia, in the Austrian Army	{ Chmelsky.
1760	Winter	Canada, among the troops in the Forts.....	Monro.
1762	Winter— Autumn	Bremen, among the English troops	Monro.
1776	Spring	Evreux, in the prison	Lepecq.
1783	Autumn	Copenhagen	Bang.
"	...	{ Jemtland, Angermanland (Sweden)	Salberg.
1784	...		
1785 } 1786 }	Winter— Summer	St. Petersburg, Cronstadt, among soldiers and in the navy; afterwards general.....	{ Guthrie, Bache- racht.

Time.		Place.	Authority.
1785 }	Spring—	Copenhagen.....	Bang.
1786 }	Summer		
1787	...	Braila	Oloff.
1789	...	Finland, among the Russian troops	Enneholm.
1793	...	Braila, during the Russian occupation	Oloff.
1798	Spring	} Copenhagen (to a moderate ex- tent	{ Bang.
1799	"		
1800	Summer	Bombay, among the English troops	McGregor, I.
1801	"	Alexandria, among the French troops	Larrey, Frank.
1803	Spring	Hungary, in the Eastern Division of Upper Hungary	Schraud.
1806	Summer	Reggio (Modena), among the French troops	Lamothe.
1807	...	Prussia, among the French troops...	
1808	Spring— Winter	Spalato, among the French troops...	Chailly.
1820	Winter	In Fort Council Bluffs (Iowa) among U. S. troops.....	{ Gale, Mower, Forry.
1822	Rainy season	Moorshedabad (Calcutta), in the Asylum for the Insane.....	Burt.
"	...	Rutnagherry (Bombay), in the prison	Bouchier.
1823	Winter	Southern Russia (Nicolajeff, Cher- son, &c.)	Lee.
1824	Winter and Spring	London (Millbank Penitentiary).....	Latham.
"	Autumn	Rangoon, among the English troops	Waddel, Ref. I.
1828-29	...	Turkey, among the Russian troops	Seidlitz.
1830	Summer	London (slight outbreak).....	McMichael.
1831	"	Prague, in House of Correction.....	Popper.
"	Spring	St. Petersburg, in House of Refuge	Doepp.
1832	...	Cannanore (Madras), among the troops	Henderson.
1833	Spring	India, throughout the west.....	Panton.
"	Autumn	Masulipatam (Madras), among the troops	Murray, I.
"	...	Rutnagherry (Bombay), in the prison	Bouchier.
1833-34	Autumn— Spring	Nusserabad (Bengal), among the troops	Ross, Mac- nab.
1836	Summer	Prague, in the House of Correction	Cejka, Popper.
"	...	England, in a number of poor-houses	Copland.
"	Spring	Iceland, in Westerland.....	Ref. II.
"	...	Adelaide (Cape Colony) among the English troops	{ Murray, II, Minto, Morgan.
1837	...	England, in a number of poor-houses	Copland.

Time.		Place.	Authority.
1837	Spring	Iceland, in Westerland.....	Ref. II.
1838	...	England, as in 1837	Copland.
"	...	Finnmark, very extensively.....	Walter.
1839	Spring	Cronstadt, in the navy	Kerewajew.
"	...	Aden, among the English troops ...	Malcolmsen.
1840	Spring	London (Milbank Penitentiary) ...	Baly.
"	...	Clairvaux, in the prison	Ref. III.
"	...	Russia, widely spread (Cronstadt, Moscow, Orenburg, &c.)	{ Samson, v. Himmel- stiern, I, II, Schütz.
"	...	Agra (N. W. Prov. of India), very extensively	{ McGregor, II.
1841	...	Algiers, in a number of localities ...	Guyon.
1842	...	Prague, in prison and garrison	Cejka, Popper.
"	Spring	Sulajew (Gov. Viatka)	Jonin.
"	Summer	Kurnaul (N. W. Prov. of India), in Military Hospital	McGregor.
1842-43	Winter— Summer	Leipzig, very extensively.....	Radius, May.
1843	Spring	Prague, general	Cejka, Popper.
1844	Spring and Summer	Christiania, in the prison	Boeck.
"	...	Alessandria, in the military prison	Novellis.
"	...	Algiers, in several districts.....	Maupin.
1845	...	Russia, in many governm.	Lingen.
"	...	Christiania	{ Same as in 1844.
"	...	Alessandria	
"	...	Algiers	
1846	Summer	Perth (Scotland), in the prison	Christison, I.
"	"	Copenhagen (frequent cases)	Ref. IV.
"	...	Christiania	{ Same as in 1884.
"	...	Algiers	
1846-47	Winter	Edinburgh, Glasgow, and other places in Scotland, very extensively	{ Christison, II, Ritchie, Ref. IV, Lonsdale, Anderson.
"	"	Exeter, Bath, Kent, Liverpool, York, Cumberland, and other places in England	{ Shapter, Laycock, Barret, Sibbald, Lonsdale, Turnbull.
"	"	In Ireland, generally.....	McCormack, Popham, Bellingham, Curran, Donovan.

Time.		Place.	Authority.
1847	Spring	Paris, in the Salpêtrière	Fauvel, I.
"	"	Givet, in the garrison	Scoutetten.
"	"	Copenhagen, slight epidemic	Hannover, Ref. IV.
"	"	St. Petersburg, very common	Lichtenstädt.
"	...	Christiania	} Same as in 1844-46.
"	...	Algiers	
1847-48	Winter— Spring	York Factory (Hudson's Bay)	Smellie.
1848	Spring	Constantinople, in workhouse	Rigler.
"	...	Algiers	Same as in 1844-47.
"	Spring	St. Petersburg, Cronstadt	Lichtenstädt, Lang.
1848-49	Winter— Summer	In Southern Russia (Bessarabia, Charkoff, Poltava, Kieff, Voronesh, Kursk, Podolia, Pensa, Volhynia, Tambov, Kasan, Jekaterinoslav, Astrakhan, Simbirsk, Viatka), generally diffused	} Ref. VII, Heinrich, Grimm, Heine, Gutteeit.
1850	...	In Melrose (Scotland), among navvies	
"	Summer	Ludwigsburg (Württemberg), in the workhouse	Ref. VIII. Dicenta.
1851	...	Fort McIntosh, and other stations of the U. S. troops in Southern Texas	Perrin.
"	Summer	Ludwigsburg (Württemberg), in the workhouse	Dicenta.
1852	...	Russia, shores of the Black Sea (Dagestan, Lesgin)	Ref. IX.
"	Summer	Ludwigsburg	Same as in 1850-51.
"	"	Rastatt, among the Austrian garri- son	Opitz.
"	...	Fort McKavit, &c., on the western frontier of Texas, among the U. S. troops	Crawford.
1853	Winter and Spring	In the Army of the Caucasus	Ref. X.
"	Summer	Ludwigsburg	Same as in 1850-52.
"	Spring	Strasburg, in the prison	Forget.
"	"	} Aix, in the lunatic asylum	Routier.
1854	"		
"	...	Wartenberg (Prussia), in the House of Correction	Wald.
"	Summer and Autumn	Breslau, in the penitentiary and the deaf-and-dumb asylum	Günsburg.
"	...	Strasburg, in the prison	Schützen- berger.
"	Spring— Autumn	Wallachia, very extensively in the Russian Army of the Danube	} Sokoloff.

Time.		Place.	Authority.
1854-56	Spring onwards	In the Crimean War, especially in the French, English, and Turkish Armies	{ Leudesdorff, Perrin, Scriver, Fauvel, Rollin, Maugin, Macleod.
1855 }	Spring	Aix, in the lunatic asylum	} Same as in 1853-54.
1856 }		In the Military Hospitals of Paris ...	
1855-56	Summer	Roanne, in the prison and beggars' refuge	Tholozan.
1856		Nicolai on the south-east coast of Siberia	
"	Summer	Ludwigsburg, in the prison	Lavirotte.
1857		Lucknow (India), during the siege...	Dawidoff.
"	Winter	Lille, in the garrison	Cless.
1860		Camp Bull, among the U. S. troops	Greenhow.
1861	...	Port Blair (Andamans), to a frightful extent among the convicts ...	Villemin.
"		In the army of the Potomac, U. S. ...	McBride.
1862	Spring	St. Petersburg, in the Obuchow Hospital	Gamack.
"	...	Prague, in the House of Correction	Herr.
1868-70		Iceland, in the fishing districts	Herrmann.
1869	Spring	Bengal, among native troops	Popper.
"	...	Paris, during the siege	Hjaltelin, I.
1870-71			Ref. XI.
1871	Winter and Spring	Ingoldstadt, among French prisoners of war	{ Delpech, Hayem, Legroux, Leven, Lasègue, Bucquoy, Boisgard, Charpentier, Georgesco, Jardin, Roche.
"	Summer	Bucharest	Döring.
1873		Prague, in the garrison hospital ...	Felix.
1873-74	Autumn—	Algiers (in Cherchell), slight epidemic among workmen	Kirchenberger.
1875	Winter	Abo (Finland), in the prison	Benech.
1875-76	Winter and Spring	Moringen, in the convict prison and in the town	Hildebrand.
1877		Paris, slight epidemic in the Mazas prison	Kühn.
			Besnier, de Beauvais.

§ 189. PRESENT AREA OF PREVALENCE.

This chronological table of all the epidemics of scurvy known to me will at the same time serve to show approximately the *geographical distribution of the disease* in recent years and at the present date. Of the 143 epidemics in the table, 35 belong to *Russia* alone, not counting the outbreaks in the Crimean War; and among these there were three, in 1840, 1845, and 1848-9, which extended over a great part of the empire. It follows, accordingly, that Russia is one of the chief seats of scurvy at the present date, although less so than in former centuries; the malady is still endemic in the Baltic provinces,¹ and in St. Petersburg,² where 2680 cases of it have been treated within the last eighteen years in the Obuchow Hospital alone.³ It is endemic also in the governments of Olonetz and of Novgorod (mostly in the circles of Beloserki, Kiriloff, Borovitsch and Tichvin⁴), along the shores of the Arctic Ocean and other parts of the Siberian littoral,⁵ such as the Amoor region (10·9 per 1000 of the troops quartered there in 1875-78 having been attacked with scurvy⁶), and in Kamtchatka.⁷ We have other accounts of its endemic prevalence in Asiatic Russia from the districts on the Chinese frontier,⁸ and from Tomsk.⁹ Also, for Russia in Europe, from the government of Kasan,¹⁰ but more especially from the southern provinces of the empire—Jekaterinoslav,¹¹ the Steppes of Saratov,¹² the Ukraine and adjoining districts of Western and Little Russia,¹³ and the Crimea.¹⁴ Mention is made also of endemic scurvy in Kutais (Trans-

¹ Krebel; Lang, l. c.

² Lichtenstädt, in 'Hecker's wissensch. Annal. der Heilkde.,' 1834, xxx, 76; Heine, 'Med.-topogr. Skizze von St. Petersburg,' St. P., 1844, Amburger.

³ Hermann.

⁴ Bardowsky.

⁵ Schrenk, Castren, 'Nordische Reise,' &c., Petersb., 1854, 271; Sollaud, 'Arch. de méd. nav.,' 1882, Juin, 435.

⁶ Seeland.

⁷ Bogorodsky.

⁸ Stubendorff, 'Med. Zeitung Russl.,' 1846, 34.

⁹ Rex, ib., 1859, 408.

¹⁰ Erdmann, 'Topogr. des Gouvernements und der Stadt Kasan,' Riga, 1822, 151, 251; Blossfeld, 'St. Petersb. Zeitschr. für Natur- und Heilkde.,' Nr. 4, 151.

¹¹ Sachs.

¹² Erdmann, 'Reisen im Innern Russlands,' Leipz., 1825, ii, 224.

¹³ Boulgakof, 'Bull. des sc. méd.,' xxiii, 205.

¹⁴ Heinrich, 'Med. Ztg. Russl.,' 1845, 379.

caucasia¹). The focus of scurvy in Southern Russia joins on, as Felix tells us, to the endemic of it in the adjoining districts of *Roumania*.

The part played by scurvy in North-Western Europe is very much less. In *Iceland*, it is true, the malady has several times broken out as a sequel of famine (last in 1836 and 1837²); but for more recent times scurvy does not deserve to be spoken of as endemic in that country;³ and the same holds good for the *Faröe*⁴ and *Shetland Islands*.⁵ As regards *Sweden*, Dalberg,⁶ writing in 1777, says that scurvy was much less common there than was generally supposed; Huss, in his medico-topographical account of the country, makes no mention of it; and Berg⁷ speaks of it as being somewhat frequent only about Umeå, the district of Udewalla, and Jemtlandslän. For the most recent period there is only one notice of scurvy in Sweden known to me, that by Heyman, referring to its prevalence in Swedish prisons; according to this there were, among 151,384 prisoners, from 1848 to 1877, 5188 cases of scurvy, or 34·3 per 1000, the percentage diminishing from 52·5 in the period of 1848-57 to 32·9 in 1858-67 and to 17·0 in 1868-77. For *Norway* there is mention (by Walter) of its somewhat frequent occurrence among the Finnish and Lapp population of Finnmarken. In *Denmark*, where it used to be rather common, it is now met with almost exclusively in prisons.⁸ The same applies to *England*, *Scotland*, and *Ireland*,⁹ where scurvy during the present century has occurred either in prisons, or in occasional epidemics, some of which were no doubt very extensive. The case is the same, too, with *Holland*¹⁰ and *Belgium* (193 cases officially reported from

¹ Krebel, *ib.*, 1858, 76.

² Holland, 'Edinb. Med. and Surg. Journ.,' 1812, April, 202, Schleisner, 'Island, &c.,' 48.

³ 'Finsen ('Jagttagelser angaaende Sygdomsforholdene i Island,' Kjöbenh., 1874, p. 56) saw only thirteen cases during a ten years' residence.

⁴ Manicus, 'Bibl. for Laeger,' 1824, Jan. 15.

⁵ Sexby, in 'Dobell's Reports,' 1871, ii, 225.

⁶ 'Tal om några det Svenska Climatats Förmåner, &c.,' Stockh., 1777.

⁷ 'Bidrag til Sveriges med. Topogr., &c.,' Stockh., 1853, 17, 22, 89.

⁸ Otto, 'Transact. of the Prov. Med. Assoc.,' 1839, vii, 211; Salomonseu, 'Udsigt over Kjöbenhavns Epidemier,' Kjöb., 1854, 126.

⁹ Curran, l. c., 109; Wylde, 'Edinb. Med. and Surg. Journ.,' 1815, July, 13.

¹⁰ Guislain, 'Annal. de la Soc. de med. de Gand.,' 1842, Jan.

1853 to 1862¹), with *Germany* and *Austria* (accounts of occasional epidemic outbreaks mostly of small extent), with *France*² and *Italy*³ (somewhat common in fortresses and prisons, especially in Venetia and the Æmilia along the valley of the Po), and with *Turkey*.⁴

From Asiatic countries we have accounts of endemic scurvy on the Yemen coast of *Arabia*,⁵ particularly in Aden ;⁶ also from some parts of *India*,⁷ such as the North-West Provinces, Rajpootana and Malwa ;⁸ from *Cochin China*,⁹ the northern part of *China*, especially Pekin¹⁰ (where it is the poor that suffer, as is usual in other countries as well), and *Japan*, where cases of a milder type (here, too, among the portionless classes¹¹) are to be seen remarkably often.¹²

The *Continent of Australia* is notorious for the severe epidemics of scurvy which have broken out time after time among the exploring parties in the interior. Pechey¹³ mentions the disease as occurring endemically, mostly among the shepherds on the Darling downs, in the north-west of

¹ Meyne, 'Topogr. méd. de la Belgique,' Bruxell., 1865, 204.

² According to Le Gendre ('Étude sur la topogr. méd. du Médoc,' Par., 1866, p. 29) scurvy is common along the hill-zone of Medoc. I have found no other accounts of scurvy being endemic in France.

³ Sormani, 'Geogr. nosol. dell' Italia,' Roma, 1881, p. 169. For the years 1874-76 the mortality from scurvy was 0·13 per 1000 among the civil population of Italy, and 0·07 per 1000 among the military.

⁴ Oppenheim, 'Ueber den Zustand der Heilkunde . . in der Türkei,' Hamb., 1833, 77; Rigler, 'Die Türkei,' &c., ii, 405.

⁵ Pruner, 'Die Krankheiten des Orients,' 334.

⁶ Malcolmsen; Courbon, 'Observ. topogr. et méd., &c.,' Par., 1861, 59. In another chapter I shall deal with the malignant ulcers which are known as Yemen or Aden sores, and which occur in many other parts of the tropics as well. Some of them, at least, are plainly of a scorbutic nature.

⁷ Particularly common in prisons (Porter, 'Madras Quart. Journ. of Med. Sc.,' 1872, Ap., p. 253). In the East Indies, according to Van Leent ('Arch. de méd. nav.,' 1867, Oct., p. 241; 1868, Sept., p. 163), scurvy is rare, being mostly found in sailors admitted into the naval hospitals. Morehead has seen it in Bombay under the same circumstances ('Clin. Res. on Diseases in India,' Lond., 1856, ii, 680).

⁸ McGregor, l. c.; Moore, 'Lancet,' 1882, June, p. 1048; Lucas, ib., Aug., p. 333.

⁹ Blanchard.

¹⁰ Morache, 'Annal. d'Hyg.' 1870, Janv., p. 54.

¹¹ Sollaud, 'Arch. de méd. nav.,' 1882, Juin, 435.

¹² Wernich, 'Geogr.-med. Studien, &c.,' Berl., 1878, 172.

¹³ 'Med. Times and Gaz.,' 1867, Nov., 509.

New South Wales. In Tasmania it is not met with as an endemic.¹ There are no accounts of it from *Oceania*.

Among the native population of the *Cape* (Hottentots), scurvy would appear to be quite unknown, according to information by Murray (II), Morgan, and Minto dating from 1836. For *Egypt*² and *Algiers* there are only a few references to epidemics (see table), and no endemic cases have been observed. In *Abyssinia*, according to Blanc,³ scurvy is found almost exclusively among foreigners, or slaves from the Shankalla country, the native population being free from it despite the use of brackish water and the want of vegetables in their food. On the other hand, in the Eastern Soudan as well as throughout the whole of the rainy zone of Africa, it would appear to be very common (especially during the overflow of the Nile) among natives and foreigners, such as travellers, hunters, camel drivers, and soldiers.⁴ On the *West Coast of Africa*, also (Benguela, Gold Coast, &c.), scurvy is mentioned as a common disease among the natives.⁵ In Senegambia it has decreased considerably of late owing to the improved hygiene.⁶

In the Western Hemisphere, the worst centres of scurvy are in the most northern latitudes—in *Greenland*,⁷ *Alaska*,⁸ and among the lumbermen of the Ottawa district (*Canada*);⁹ in these cases the disease is nearly always among the strangers, the natives enjoying an almost complete immunity.¹⁰ In the *United States of America* scurvy to any considerable extent has not been seen in recent times except

¹ Hall, 'Transact. of the Epidemiol. Soc.,' 1865, ii, 85.

² Pruner, l. c.

³ 'Brit. Med. Journ.,' 1869, March, 278.

⁴ Hartmann, l. c.

⁵ Magyar, 'Reisen in Süd.-Afrika,' &c., from the Hungarian, Pesth, 1859, i, 450; Clarke, 'Transact. of the Epidemiol. Soc.,' 1860, i, 107; Chassanil, 'Arch. de méd. nav.,' 1865, Mai, 508.

⁶ Borius, ib., 1882, Mai, 371.

⁷ Lange, 'Bemaerkn. om Grönlands Sygdomsforhold,' Kjöbenh., 1864, 28.

⁸ Blaschke, 'Topogr. med. port. Novi-Archangelensis,' Petropoli, 1842, 67.

⁹ Grant, 'Med. Times and Gaz.,' 1863, Dec.

¹⁰ Lange writes in this sense as regards Greenland. Blaschke says that he did not see a single case among the Kolosks and Aleutians during a residence of several years at New Archangel. Gras says that it is quite unknown in the small island of Miquelon, Newfoundland ('Quelques mots sur Miquelon,' Montp., 1867, p. 39).

among bodies of troops who had been exposed to great privations during the war or quartered at outlying stations;¹ also at the time of the "gold rush" in California² among immigrants who suffered in much the same way. In the *West Indies* it appears that on some of the islands it is very rare. Lemprière,³ after six years' observation of the diseases of Jamaica (1792-97), gives it as seldom occurring there; in Martinique, according to Rufz,⁴ it is quite unknown. As against these statements we have the assertion of Levacher⁵ that scurvy is prevalent in the Antilles to a great extent; but the silence of other observers in those regions makes it impossible to decide how far that is true only for particular localities.

From *South America* there are no accounts relating to scurvy that are at all trustworthy. Sigaud⁶ mentions it as occurring in *Brazil* among newly imported negroes; but from a later account it would appear that the malady had been already developed among these unfortunates during the passage.

§ 190. OUTBREAKS AT SEA.

Particularly interesting for the etiological inquiry concerning scurvy are the observations of recent date on the *epidemic occurrence of the disease on board ships*; inasmuch as they present an easily surveyed and obvious field of observation, and therein furnish valuable materials for judging of the circumstances that determine the existence of the malady, or at any rate are likely to favour it.

Scurvy at sea has become much rarer since the end of

¹ Hammond ('Amer. Journ. of Med. Sc.,' 1853, Jan., 102), for the Mexican war; Perin and Crawford (ll. cc.), for several forts in the west of Texas; Madison ('Statist. Reports on the Sickness and Mortality of the U. S. Army, 1855-60,' Wash., 1861, 40), for Fort Randall (Dakota Terr.); Johns (ib., 45), for Fort Laramie (Nebraska Terr.); Bartholow ('Amer. Journ. of Med. Sc.,' 1860, April, 330), for Fort Bridger (Utah Terr.).

² Logan, in 'Southern Med. Reports,' ii, 468.

³ 'Pract. Observ. on the Diseases . . in Jamaica, &c.,' Lond., 1799, i 50.

⁴ 'Arch. de méd. nav.,' 1869, Nov., 349.

⁵ 'Guide méd. des Antilles,' Par., 1840, 145.

⁶ 'Du climat et des malad. du Brésil,' Par., 1844, 133

last century, the period when the hygiene of ships underwent great improvement, the provisioning of ships more particularly being looked after with all possible care as regards adequacy and suitability, especially in the case of long voyages. It is hardly ever met with now except where unforeseen difficulties in suitably providing for a ship's company arise through misadventure. In these reforms England has set a brilliant example to other seafaring countries. In 1795 there were introduced into the English navy those admirable regulations for provisioning ships of war which Blane drew up; and since that time scurvy has been so rare with them that the number of cases over the whole fleet during the years 1856-61 did not amount to more than 1.05 per 1000 men.¹ It is on the Australian Station and on the West Coast of Africa² that the disease is oftenest seen.

The following are the more considerable outbreaks of scurvy in ships of the English navy that have been recorded during the last fifty years: 1838 in the *Palinurus*³ cruising on the North-East Coast of Africa; 1839 in the *Alligator*,⁴ ordered from England to the North Coast of Australia, the disease having broken out four months after sailing from the home port; 1854 in the British fleet in the Black Sea⁵ during the Crimean War; same year in a troopship⁶ on the voyage to India; and 1866, a severe epidemic among the soldiers on board a troopship⁷ returning from India, the vessel being overcrowded as well as short of provisions.

Until not very long ago the state of matters was less satisfactory in the ships of the British mercantile marine, particularly in those sailing to or from ports east of the Cape of Good Hope, which had to be long at sea, the first cases of scurvy occurring when they were sixty to eighty days out. During the period from 1852 to 1863, the cases of sickness of all sorts in the English mercantile marine reported to the authorities amounted to 25,486; and of

¹ Friedel, 'Die Krankheiten in der Marine,' Berlin, 1866, 271.

² Bryson, 'Ophthalm. Hosp. Reports,' 1859, July.

³ Hardy, 'Transact. of the Bombay Med. Soc.,' 1839, ii, 256.

⁴ See 'Statist. Report on the Health of the Navy, 1837-43,' Lond., 1853, pt. ii, 21.

⁵ Rees, 'Med. Times and Gaz.,' 1854, Sept., 233.

⁶ Morgan, ib., Dec., 586.

⁷ Wrench, ib., 1867, March, 317.

these 1058 or 4·2 per cent. were cases of scurvy. Of 372 cases of the disease admitted from merchant ships into the Seamen's Hospital, Greenwich, from 1863 to 1866, 316 belonged to vessels that had sailed from ports east of the Cape. One reason of this comparatively frequent occurrence of scurvy in the mercantile marine is undoubtedly carelessness in the provisioning of the ships for the voyage, the blame resting not unfrequently with the captain. Another reason is the difficulty of procuring fresh provisions during the voyage; in which connexion attention has been called to the state of matters at the port of Aden, where fresh vegetables were particularly hard to get.¹

The care with which the English Government has endeavoured to cope with scurvy on shipboard since the beginning of the century has been emulated by the other seafaring countries of Europe and America; and since that time the malady in their ships also, and especially in their ships of war, has been considerably lessened. In the Austrian navy the cases of scurvy from 1863 to 1870 were 1 per cent. of the ships' companies; in 1871 and 1872 they had fallen to 0·34 per cent.² In the German navy from April 1875, to March 1880, there were only sixteen cases of fully developed scurvy, besides seventy-six cases of scorbutic affection of the gums;³ taking the cases of both kinds together, the sick-rate was 0·475 per cent.; and in only three ships did the malady attain to slight epidemic diffusion.

I am acquainted with only a few casual notices of scurvy on board ships of the Dutch,⁴ Italian⁵ and United States⁶

¹ See 'Brit. Med. Journ.,' 1867, Jan., 89, Feb., 147; 'Lancet,' 1867, April, 463; and Dickson, 'Transact. of the Epidemiol. Soc.,' 1867, ii, 440.

² 'Statist. Sanitätsbericht der Kaiserl. öster. Kriegsmarine für das Jahr 1872,' Wien, 1874, 26.

³ 'Statistische Sanitätsberichte der Kaiserl. deutschen Marine vom April, 1875, bis März, 1880.'

⁴ Lilienfeld (in 'Casper's Wochenschr. für Heilkde.,' 1851, Nr. 1-3) gives an account of an outbreak in 1849 on board a Dutch man-of-war, ordered from Batavia to China and California, after her arrival in the bay of San Francisco.

⁵ Report by Vieira ('Revist. med. flumin.,' 1838, p. 318) on the scurvy on board the Sardinian frigate *Euridice* in 1836 during a voyage from Para to Rio de Janeiro.

⁶ See Coale ('Amer. Journ. of Med. Sc.,' 1832, Jan.), on the epidemic of scurvy which broke out in a United States' frigate off the coast of China during a voyage round the world; also Foltz (ib., 1848, Jan., p. 38), on the epidemic of 1846 in the flotilla in the Gulf of Mexico.

navies. In the French ships-of-war it has been epidemic comparatively often.

Thus in 1827-28, in the Mediterranean blockading squadron;¹ 1842, on board the frigate *Heroïne*² cruising on the coast of New Zealand; 1846-47, in the *Belle Poule*³ cruising between Bourbon and Madagascar; 1854-55, in the French squadron in the Black Sea during the Crimean War (so severe that the strength of the ships' companies was quickly reduced by scurvy to one half);⁴ 1859, on board a ship-of-war on the passage from France to China, the disease appearing when they were 120 days out and invaliding 230 out of 716 persons on board;⁵ 1864, in the frigate *Andromaque*,⁶ also from France to China; same year, in a corvette⁷ cruising in the Gulf of Mexico (Bay of Matamoras); same year, in the frigate *Néréide*⁸ on a voyage round the world; 1867, in a frigate⁹ from Mexico to France; same year, in a ship-of-war¹⁰ on the voyage back to France after a four months' cruise round Iceland. There have also been very severe epidemics of scurvy on board ships of the French navy employed in transporting convicts to New Caledonia: namely, in the frigates *Sibylle*¹¹ and *Iphigénie*¹² in 1866-67, and in the *Orne*¹³ and *Var*¹⁴ in 1873.

Lastly, particular attention is due to the very severe outbreaks of scurvy that have been often observed on board whalers which have had to endure the want of provisions, especially fresh vegetables, owing to their being long beset in the ice or kept back by contrary winds.¹⁵ Under the

¹ Levicaire, 'Gaz. méd. de Paris,' 1832, 735.

² Dutroulau, *ib.*, 1850, 627.

³ *Id.*, *ibid.*

⁴ Arnaud, 'Gaz. méd. d'Orient,' 1857, Juin; Beuzelin (see Bibliogr.).

⁵ Lagarde, *l. c.* (Bibliogr.).

⁶ Lajartre, 'Considér. sur l'état sanitaire de la frégate l'*Andromaque*, &c.,' Par., 1866.

⁷ Pirion, 'Arch. de méd. nav.,' 1865, Nov., 415.

⁸ Bernès-Lasserre, 'Rélat. méd. de la campagne de la frégate la *Néréide*,' Par., 1866, 21. The reader will be astonished to find from this report that there were cases of scurvy on board immediately after the ship left Brest.

⁹ Léon, 'Arch. de méd. nav.,' 1868, Avr., 290.

¹⁰ Galliot, *ib.*, 1877, Mai, Juin.

¹¹ Normand (see Bibliogr.).

¹² Caurant (*l. c.*).

¹³ Ayme (*l. c.*).

¹⁴ Ledrain (*l. c.*).

¹⁵ Instances within recent times are given by Williamson ('Lond. Med. Gaz.,' 1836, April, xviii, 136) and Smith ('Edinb. Med. Journ.,' 1868, March, 859), for Hudson's Bay. A French authority ('Arch. de méd. nav.,' 1867, Mai, p. 274) speaks of the numerous cases of scurvy that have been admitted into the hospital at St. Pierre (Newfoundland) in Frenchmen engaged in the whaling, or in

same circumstances scurvy has become epidemic among the crews of ships engaged in Arctic discovery; as in the *Investigator*¹ in 1852, the malady appearing after the vessel had been two years and three months out; and in the *Alert* and *Discovery* (under Nares) in 1876.²

This sketch of the history of scurvy and of its geographical distribution at the present time can lay no claim to completeness; on the one hand there are doubtless many records that have escaped my notice; and, on the other hand, many persons who have observed epidemics of scurvy have neglected to report them. But the most serious gaps in our knowledge of the incidence of scurvy pertain to those regions which are somewhat remote from ordinary traffic and whose general health-conditions have been very deficiently observed and recorded. So far as our information goes, we may at least conclude that scurvy, although no longer so common as in past centuries, can by no means be said to be extinct; on the contrary, that it still threatens to break out among bodies of people the moment they begin to experience those hardships and mishaps of living which I shall have to deal with in the paragraphs following.

§ 191. QUESTION OF THE INFLUENCE OF COLD AND WET.

The records of scurvy both in former centuries and in recent times teach us that the malady is not dependent for its existence and extension on any particular *climate*, or on the geographical position of the place or country; but that it has been found all over the world, equally in temperate latitudes and in the coldest and hottest. No doubt when we compare the amount of scorbutic sickness in various parts of the world, we shall find that there is a small excess for

working at the cod-liver oil manufacture. Under similar circumstances the disease has often appeared, according to Lallemand ('Casper's Wochenschr. für die ges. Heilkde.', 1848, pp. 25, 385), among the crews of guano ships, particularly when exposed to extreme want after shipwreck on the Patagonian coast.

¹ Armstrong, l. c., 13, 36.

² Donnet and Fraser (Bibliogr.); see also Leach ('St. Barth. Hosp. Rep.', 1878, xiii); and Rochefort ('Arch. de méd. nav.', 1877, Juill, Aout, Sept.), following Donnet.

regions within the cold zone ; and that fact, taken along with a number of observations on the season of prevalence of the malady, has given rise to the often expressed opinion that *states of the weather*, particularly cold and wet weather, are a principal factor in the production of the disease, and that its maximum falls, accordingly, in the *seasons* to which such states of weather mostly correspond. The following analysis should show how far this opinion is justified by the facts :

Of 73 epidemics of scurvy in cold and temperate latitudes, of which the date is given with tolerable precision, 24 occurred (or reached their highest point) in winter, 34 in spring, 13 in summer, and 2 in autumn. Of 10 epidemics in tropical and sub-tropical regions, 1 falls in the cold season, while 4 fall in the hot and 5 in the transition-seasons corresponding to our spring and autumn. Accordingly, for the cold and temperate zone, spring would be the proper season of scurvy, and next to it would come winter. In agreement with Stoll¹ who says that “ *constatissima causa (scorbuti) est mador atmosphaerae continuus, maxime frigidus,*” many who have observed² these winter and spring epidemics or endemics have laid special stress for the pathogenesis upon the cold and wet weather which had been prevalent at the same time as the disease. In like manner the outbreaks of scurvy on board ship have been traced in many quarters to the direct influence of the weather. Such was the view that Lind very decidedly took ; he pointed to the kind of life led by the common sailors, to the frequent drenchings of their clothes and their fore-castle, and to the dampness of their sleeping-bunks. He then contrasts the exemption from scurvy that the officers in most cases enjoyed, explaining it by their being less exposed to hardships or better protected against them. A similar opinion was held by Rouppe,³ who dwelt upon the fact that scurvy usually breaks out on board a ship as she passes from the tropics to higher latitudes ; and by Macmichael, Pirion and others. But, against these observations adduced in favour of the in-

¹ ‘ *Praelect. in diversos morbos chronicos,*’ ed. Eyerel, i, 7.

² Bacheracht, Nitzsch, Monro, Radius and May, Curran, Routier, Opitz, Perrin, Jouin, Heinrichs.

³ ‘ *De morbis navigantium,*’ Lugd. Batav., 1764, 118.

fluence of cold and wet, there is a not less extensive series of well-authenticated facts which afford evidence, both of a negative and positive kind, that this factor plays only a secondary part in the production of the disease, if indeed it come into account at all. All the localised epidemics of scurvy mentioned by Lavirotte in the penal institution at Roanne, by Dicenta and Cless in the prison of Ludwigsburg, and by Popper and Kirchberger at Prague, occurred in summer. Chrastina¹ observes that, in the Charitable Institution at Alserbach (Vienna), where there is scurvy every year, a few cases occur as early as February and March, but that the number increases in the months following, and reaches the highest point in July. Of the 2680 cases of scurvy observed at the Obuchoff Hospital of St. Petersburg during eighteen years, the percentages in the several months were as follows (according to Herrmann) :

January . . .	3'06	May . . .	21'86	September . .	1'97
February . . .	5'07	June . . .	20'55	October . . .	1'52
March . . .	9'32	July . . .	14'70	November . .	1'49
April . . .	12'91	August . . .	5'26	December . .	2'23

Here the maximum is in May and June, and the minimum from October to December. Amburger arrived at nearly the same result on comparing the states of the weather at St. Petersburg for each month from 1867 to 1880 with the number of scorbutic cases admitted into the hospitals of the city : "The evidence is plain," he says, "that scurvy is not dependent on meteorological influences." Almost all the epidemics in Swedish prisons from 1848 to 1877, according to Heymann, have begun in summer, and mostly towards the end of it, and have terminated about the close of the year ; in these cases the influence of cold and wet weather on the pathogenesis is absolutely excluded. From the reports of the army surgeons on the prevalence of scurvy among the French troops during the Crimean war, we find that the monthly returns of the sickness from October 1854 to June 1856, including a total of 23,365 cases, are as follows :

¹ 'Oest. Zeitschr. für Heilkde.,' 1859, Nr. 12.

1854	October	.	.	20	1855	September	.	.	1388
	November	.	.	80		October	.	.	707
	December	.	.	800		November	.	.	718
1855	January	.	.	1575		December	.	.	1248
	February	.	.	789	1856	January	.	.	3980
	March	.	.	452		February	.	.	4341
	April	.	.	348		March	.	.	1787
	May	.	.	132		April	.	.	785
	June	.	.	350		May	.	.	275
	July	.	.	1140		June	.	.	50
	August	.	.	2400					

According to this, the epidemic reached its first maximum in the summer of 1855 and its second maximum in the winter of 1855-56. From tropical and sub-tropical regions there are other observations in entire agreement with these facts. In the Indian gaols the epidemics of scurvy reach their greatest height in the months of July, August, and September (Porter);¹ and McGregor confirms this when he tells us that the disease is most inveterate in the North-West Provinces during the hot season, having been so in 1839 at Agra and in the hospital at Karnaul, where the air was at the same time very dry.

Wernich says that in Japan scurvy is most commonly seen in summer. Logan, after describing the kind of weather at the time when scurvy broke out among the gold diggers in California, goes on to say: "If the facts here given have any value at all, they should certainly serve to prove that cold and wet, which have hitherto been regarded as the two most powerful predisposing causes of scurvy, were altogether unconcerned in producing the malady in this country."

In a much more equivocal light than in the case of land scurvy, does this etiological factor exhibit itself in connexion with scurvy on board ship. Rouppe's assertion that it develops for the most part as the vessels pass from low latitudes into higher, is altogether unfounded. If we are to have a general rule of that sort at all, it would be much more correct to express it in exactly the converse way; for, as Bampfield² had previously pointed out, scurvy on board ship has occurred much more often in the tropics than in

¹ 'Madras Monthly Journ. of Med. Sc.,' 1872, April, p. 253.

² 'Treatise on Tropical Dysentery,' Lond., 1823, p. 239.

higher latitudes. We have, besides, the evidence of numerous authorities such as Léon, Lascade, Lejartre and others, based upon the systematic observations made during long voyages (from France to the East Indies or the Pacific), that scurvy occurs on board ship altogether independently of meteorological influences. The slight dependence of the malady under given circumstances on the coldness of the season, is further shown in the statistics published by Duchek¹ of the sick-rate from scurvy in the Austrian Navy, the same having been 0·09 per cent. of the full strength in winter, 0·13 in spring, 0·17 in summer, and 0·08 in autumn.

Again, the opinion sometimes expressed that the outbreaks of scurvy, which used certainly to be common in former times in ships making the voyage round Cape Horn, were to be attributed to the prevailing bad weather, is shown by an interesting observation of Logan² to have little general relevancy. In the worst season of the year they were beating about for four months in a small schooner trying to double Cape Horn; during all that time the crew were never out of their wet clothes, the ship with everything in it was saturated with water, the cabins had to be kept shut and were accordingly full of all kinds of emanations; and yet not a single trace of scurvy showed itself on board.³ It is not the fact of sailing in high or low latitudes, and not one season more than other, but solely the length of the voyage in relation to the store of suitable provisions, that really gives rise to an outbreak of scurvy on board ship. And this is the explanation, as we shall afterwards see, of the material decrease of scurvy at sea in recent times, although ships' crews are liable as much as ever to have the weather both foul and fair.

In the etiology of scurvy, diet is the Alpha and Omega; but inasmuch as weather and season do come into play directly or indirectly, we must think of these factors as

¹ L. c., p. 279.

² 'Southern Med. Reports,' ii, 474.

³ [In the voyage made classical by Dana in his 'Two Years before the Mast,' the experience of Cape Horn weather was much the same, only less protracted. The single case of scurvy which he mentions (noteworthy also for the almost miraculously quick cure by the juice of raw potato) occurred when the ship was in the warmer latitudes.]

always able to exert a modifying influence on the disease in one direction or another. Moreover, it cannot be denied that weather-influences which lower the resistance power of the organism by disturbing the normal course of its functions, may predispose it to an attack of scurvy; but this applies not only to cold and wet weather, but also to the relaxing effects of a prolonged residence in a warm temperature, as Foltz and other observers have pointed out.

§ 192. THE KIND OF SOIL OF NO ACCOUNT.

Again the significance of the kind of soil for the occurrence of the scurvy must be judged in this and no other way. The exceptionally frequent prevalence of the disease at sea is itself an evidence that its production is altogether independent of strictly telluric influences; and any direct influence of the soil upon the pathogenesis is excluded not less by the very narrow limitation of epidemics, especially in recent times (often to a few rooms), as well as by the material decrease of the malady under the blessings of rational hygiene, and, above all, of sufficient and wholesome food. A number of observers have attached special importance to the morbid effects of a wet soil in scurvy; such as Oloff for the epidemics in Wallachia, Monro for Bremen, Scoutetten for Givet, Günsburg for Breslau, Perrin for the Crimea, Novellis for Alessandria, Opitz for Rastatt, Döring for Ingolstadt, Blanchard for Cochín China, and Seeland for the Amoor region. But the conclusion that they draw from the facts is valid only in so far as relates to a possibly injurious effect of residence on a damp soil, or to an increased personal predisposition to scurvy thereby caused. It is the more necessary to be cautious in judging of these circumstances in the causation, for the reason that many other harmful things besides a wet soil come into account in all such cases, most of all the food. When Grimm alleges "that territories traversed by great rivers, or situated on the estuaries of rivers, or on the sea-coast, have a visitation of scurvy every spring, whenever there has been a cold and wet winter preceding," he commits an inexcusable exaggeration and a gross error, inasmuch as

scurvy has been as often upon dry ground as on wet, on elevated land as on low levels, on rocky soil as on porous and pervious. Lastly, when some observers (including Seeland among recent writers) speak of a scorbutic miasma developing from the wet ground, and would class the malady among infective and even communicable or contagious diseases (a point to which I shall recur), I can see nothing more in this than a misdirection of the etiological inquiry, of the same kind that has found abundant expression lately in the speculative theorising and in the rage for parasites which have taken possession of the medical world.

§ 193. INFLUENCE OF OVERCROWDING AND OF IMPURE AIR.

There is one factor in the history of scurvy which has been uppermost in every age and in all countries, namely, the close association of the disease with *defects of hygiene and above all of diet*; and that factor must in reason become the point of departure in the inquiry into causes.

In the first place, we meet in this connexion with the noteworthy fact that of the 144 epidemics on land in the above table, only 42 were general, and of these 42 only 26 extended to more than one locality and very few to large tracts of country. On the other hand, there were 55 observed in besieged fortresses, or in the besieging armies,¹ or in garrisons; and 47 in prisons, poor-houses, and refuges, or under such like circumstances. In all the cases where scurvy has appeared outside these circumscribed areas, it is only the needy part of the population, wanting the barest necessities of life, and in particular a sufficiency of proper food, that have suffered; the better off classes have been altogether exempt or have been touched by the disease only in so far as the same wants have made themselves felt among them also. This applies to the incidence of the disease among bodies of troops both on shore and afloat; for it is usually only the common soldiers or sailors and the petty officers that suffer, while cases among the officers have been the exception and

¹ Olaus Magnus says of scurvy, "*Est morbus castrensis qui vexat obsessos et inclusos.*"

have occurred only under such exceptional circumstances, (particularly in Arctic expeditions) as made the privations general.¹

It stands to reason that in all these cases we are concerned with an aggregate of influences injurious to health. The question then arises whether the disease ensues as a consequence of their combined effect, or proceeds from influences of one particular category. In framing an answer to this question we must above all bear in mind that the misery of living, with its consequences, has at all times existed over the whole world; whereas scurvy as an epidemic malady has been a phenomenon of comparatively rare occurrence, and as an endemic has played a very subordinate part, especially in recent times; and accordingly that the noxious thing which has arisen out of the hardships of living and has become the proper cause of the malady, must have had something of a specific character in it.

It has been thought probable that *the tainted atmosphere caused by overcrowding* may have been a material factor in the etiology, perhaps the one decisive thing; some observers have even gone so far as to allege that a specific scorbutic miasma has developed under these circumstances. This conjecture supports itself on the fact that there has not unfrequently been overcrowding at the time of an outbreak of scurvy in one of those self-contained institutions before mentioned; and that on board ship, especially in transports, scurvy has attacked those of the crew, or of the troops or convicts, who have passed the greater part of the time, or perhaps in bad weather the whole of the time, in the overcrowded and insufficiently ventilated 'tween decks or lower hold. One cannot, *a priori*, deny that close confinement in an atmosphere of that kind, little suited as it is to the needs of respiration, and the absence of exercise in the open air, are

¹ The following instances are recorded: by Kramer, for the Imperial army in Hungary 1734-35, when none but the common soldiers were affected; by Monro, for the English troops in Bremen in 1762, when not merely the officers and staff escaped, but also the sergeants; by Forry, for the epidemic among the United States' troops at Council Bluffs in 1820, when only one officer took scurvy; by Coale, for the U.S. frigate *Columbia* on a voyage round the world, only three out of the twenty-eight officers being affected, and these the officers who had shared in the diet of the scurvy-stricken crew.

not without their effect on the composition of the blood and the nutrition of the body, and may accordingly become a predisposing cause of an attack of scurvy.

The following remarks by Armstrong¹ on the influence of foul air upon the condition of patients with scurvy, are selected from a large number of observations to the same effect: "It is impossible to deny some degree of influence to the effects of pure air in this disease. I have found, where the ventilation was occasionally rendered less perfect than usual for a few days (from unavoidable causes), and the escape of impure air was thereby interfered with, that those who were labouring under the disease always experienced more or less aggravation of their symptoms. I think that the existence of a vitiated atmosphere always tends to impart a more aggravated character to scurvy. Hence, attention to ventilation in this disease, as in every other, should always be an object of our greatest care."

But we shall keep the importance of this noxious agent within due limits when we reflect that the outbreak of scurvy in places of confinement, especially prisons, poor-houses, and the like, has never been more than an occasional incident notwithstanding their incessant overcrowding; and that in some instances, as, for example, the convict prison at Ludwigsburg, the disease has been prevalent year after year altogether irrespective of the greater or smaller number of inmates; or that, in one and the same place, there have been two centres of disease (as in 1854 at Breslau, in the House of Correction, and the deaf-and-dumb institution) of which one had been overcrowded, and the other not only not so but even admirably ventilated and a pattern of cleanliness. We have to bear in mind, also, that epidemics of scurvy have broken out in prisons where there could be no question whatsoever of overcrowding, as at Roanne in 1856 (Lavirotte), Exeter in 1847 (Shapter), the Paris House of Correction in 1871 (Delpech, Georgesco), as well as very often on board ship; and that the malady has several times decimated the expeditions sent out to explore the interior of Australia, breaking out, that is to say, among persons living constantly in the open air. It has further to be said that most of the observers who are inclined to lay special stress on overcrowding, cannot but admit that other hardships of living, and most of all a deficient diet, are contributories in the pathogenesis. It is clear then, that this factor, like the bad weather and wet soil already mentioned, and like bodily fatigue, thwarted feelings, such as home-sickness, and other things that some writers have specially dwelt upon in the causation, has no other importance than as a *causa prædisponens*.

¹ L. c., p. 32.

§ 194. CAUSED BY WANT OF FRESH VEGETABLES.

No single one of these things, nor the whole group of them together, has ever yet given rise to an epidemic or endemic of scurvy. There must always have been at the same time in action one particular noxious influence, an influence which has produced scurvy quite independently of these, and not unfrequently under hygienic conditions that were in other respects as good as can be conceived—I mean *something wanting in the food*. It is in that then, that we must seek for the most material if not the exclusive cause of scurvy. There are few points of etiological doctrine about which so much agreement has been expressed among observers of every period and in all countries, as the power of an inadequate diet to determine an outbreak of scurvy; although opinions differ as to whether it is want of food in general, or the absence of certain elements of the food, chiefly of a vegetable kind, or a monotonous diet limited to one kind of nutriment, especially salt meat, or finally something positively harmful or unsound in the food or drink, which furnishes the real cause of the malady.

Against the notion of *deficiency of food in general* (irrespective of the quality) being of itself productive of scurvy, there is first of all the fact—and to my thinking the unanswerable fact—that the severest of famines, due to failure of the crops or other elemental causes, or to the calamities of war, have only in very rare instances been attended by or followed by epidemics of scurvy; that the great and proper types of famine-sickness, as seen of late in Algiers, India, and other parts of the world, have never been the scorbutic, and that scurvy is as far as possible from bearing the stamp of a disease of inanition. Conversely, and still telling against the same notion or at least against the general application of it, we have the fact that in very many instances, both on sea and land and among people living in the open air as well as those in confinement, scurvy has broken out although there has been no absolute want of food at the time. No doubt there have been some epidemics of scurvy in consequence of bad harvests or famines from other

causes: as in Sweden in 1784, at St. Petersburg, Cronstadt and other places in Russia in 1785, Braila in 1787 and 1793, Hungary in 1803, Southern Russia in 1823, 1845, and 1849-50, Iceland in 1836, Moscow in 1840, Prague and Leipzig in 1843, Great Britain and Ireland in 1846-47, and Paris in 1871. But for most of these cases it can be proved, as we shall see in the sequel, that it was not the want of nutritive articles absolutely, but of a certain kind of them, that occasioned the sickness.

There is equally little justification for the idea that it is the immoderate or almost exclusive use of salt meat which creates the real occasion of the malady. Much of the evidence that passes current in support of this rests upon a loose sort of criticism; inasmuch as it is always a question when scurvy breaks out under these circumstances, whether the disease is indeed the effect of that kind of diet, and not rather of the want of other nutritive substances, namely, vegetables. Further, there have been a number of outbreaks of scurvy observed, in which the affected persons had certainly suffered from no lack of fresh meat: as in the Imperial army in Hungary in 1735; in Lord Anson's fleet in 1741, which left the Mexican coast provisioned with fresh meat; at Millbank Penitentiary, London, in 1823; among the English troops at the Cape in 1836; among the Austrian troops at Rastatt in 1852; in Ireland in 1846 (Curran); in Burke's expedition through the heart of Australia in 1861; in Paris and among the French prisoners at Ingolstadt in 1871. Lastly, against any scorbutic effect due to a full diet of salted meat or fish, there is the fact that many of the peoples of the far north, such as the Finns, Lapps, Koriaks, Tchoukchi, and Eskimo, will subsist often for months on pickled flesh or salted fish without having any visitation of scurvy, or at the most only one now and then.

Beckler, in his interesting account of the outbreak of scurvy among the members of Burke's exploring party in the interior of Australia, points out that the provisions which the expedition took with them from Melbourne were unexceptionable, and that scurvy appeared, none the less, when they were obliged for want of fresh water, to drink the standing water in the half-dried creeks, fouled by worms

and weeds. Beckler is all the more disposed to regard that as the true and only cause of the malady, for the reason that the expedition which was sent out subsequently to bring back the scurvy-stricken explorers from the interior to the Darling river, and which took with them only such provisions as remained over from the outfit of the original party, continued free from the disease; the reason being, as Beckler observes, that they were so fortunate as to obtain pure water from the rains that had meanwhile fallen in the interior. Well founded as this conclusion of Beckler's may at first sight appear, there are some very considerable objections to it, as we shall see in the sequel. Furthermore, it can by no means be denied that a monotonous and innutritious diet (salted meats and fish being in the first rank of such) or unsound food, may act on the organism just like absolute want of food, so as to lower its powers of resistance and thereby increase its predisposition towards the attack of sickness.

The central point of interest in the problem of the real cause of scurvy is undoubtedly how far the *want of fresh vegetable food*, or rather of a particular kind of it, is of paramount importance in the pathogenesis. Even if this notion were not supported by an overwhelming body of experience as to the usual circumstances under which it has always appeared in all parts of the world both ashore and afloat, among those leading a free life as well as among those living under some restraint, it would be sufficiently established by the one fact that there is no more certain means of obviating an outbreak of scurvy, and no more speedy means of suppressing it, than a diet of fresh vegetables of the right kind, or the medicinal use of their principles.

The importance of this matter, and the doubts about it which are still raised here and there, will justify me in giving briefly a few of the more remarkable among many experiences that have been collected to illustrate the point.

The first to express an opinion as to the morbid influence of a want of fresh vegetables was Bachstrom,¹ who thus summarises his experience in the matter during the epidemic of scurvy at Thorn in 1703:

"Probe itaque pensitatis omnibus circumstantiis, quas superius in

¹ 'Observ. circa scorbutum,' Lugd. Bat., 1734, p. 95.

historia et cura hujus morbi narravimus, imo pluribus aliis quas hic ob brevitatem omittimus, concludendum nobis esse videtur: causam veram et primariam scorbuti nullam aliam esse, quam abstinentiam diuturniorem a quocumque genere recentium vegetabilium, sive illa climatis indoli, sive coactioni et necessitati, sive neglectui sit tribuenda."

The epidemic of scurvy in Hungary, in 1734, led Kramer¹ to the same conclusion; he noticed that it was almost always the common soldiers, and not the officers, who sickened; the reason being that "the former had nothing but farinaceous substances and legumes to eat, while the latter had often green esculent vegetables; when some of the officers neglected these, which did not happen often, they took scurvy also." Monroe,² in his account of the scurvy in Canada, in 1760, and at Bremen, in 1762, says that the disease is most frequent in those northern countries where fresh vegetables are scarce, and the inhabitants obliged to live much on salted provisions during the winter. That was the reason why the malady was so common in Quebec during the first winter of its occupation by the English, as well as in several other forts in America which were captured so late in the season that the troops had no time to lay in a store of vegetables and fresh meat, and were obliged to live mostly on ships' stores. At Bremen the disease was seen among the soldiers only, the reason being that they (although not the officers, civilians and others) were unable to procure vegetables or fruit in the market. In reference to the epidemic of scurvy in Hungary in 1803, Schraud lays most stress for the etiology upon the want of food, and especially of fresh vegetables, during the winter. Still more definite is Guthrie's opinion as to the cause of the epidemic of 1785, among the naval and military forces at St. Petersburg and Cronstadt. The cause was found to be the want of those vegetables upon which the maintenance of health depends; cabbage, carrots, beet and turnips ran short, and the small supply sent the price up much too high for the pockets of the troops. It was just among them that the disease became prevalent; while the labouring class and the country people understood better how to provide for themselves. Lee, in his account of the severe epidemic of 1823 in Southern Russia, says, with reference to the laying waste of the fields by swarms of locusts, "It was precisely at this period that the disease commenced, and there can be little doubt that it ought to be attributed to a want of the usual supply of the sour crout and prepared cabbages and other vegetables for the winter . . . All green vegetables had attained an unusual price at this period, and it was in the power of few to make the usual provision. In all the hospitals the patients were supplied, as they usually are, with fresh animal food, bread and grits for gruel, yet the disease was not arrested."

Under the same circumstances of a scarcity of fresh vegetables, the malady became prevalent in 1840, in Orenburg (Schütz) and in Moscow (Samson v. Himmelstiern, 2nd ref.); also in 1842 in Viatka, where the

¹ L. c., p. 80.

L. c., p. 204.

real cause, as alleged by Jonin, was "the absolute want of the kind of vegetables that are indispensable for preserving the right composition of the body's juices;" again, in 1854 among the Turkish and French troops in the Crimean War, while the English army suffered little, having better provisions. Leidesdorf remarks, in his account of the scurvy in the Turkish army, that those who suffered most were the Tartar regular cavalry, whose food was often only a few handfuls of rice, some rancid fat and mouldy biscuit, eked out, at the best, with damaged beans or peas and salted mutton, while of fresh vegetables there were absolutely none. Regarding the outbreak among the French troops also, Perrin, Fauvel, Rollin, and most of all Scrive,¹ lay stress chiefly on the entire want of vegetables, of which there are never any in that region during the winter, while all the esculent plants of the summer season had been burned up around the camp before Sebastopol by the excessive heat. The epidemic of scurvy at Prague in 1843 is referred by Cejka to the failure of the harvest the year before; vegetable growth of every kind was poor, but potatoes in particular were deficient both in quantity and quality; so that not merely the soldiers and the poor, but even the residents in better circumstances, had to put up for a long time with bread and coarse pastry.

There is absolute unanimity among the English, Irish, and Scotch practitioners, that the epidemic of scurvy in the United Kingdom during 1846-47 was due to the bad harvest, and particularly to the failure of the potato crop. Lonsdale's conclusions, from the observed facts during the epidemic in the South of Scotland are: "That scurvy originates from an error of diet,—the occupation, dwellings, &c., sometimes viewed as collateral causes having little or no influence,—and that a deficiency of potatoes constitutes the chief error of diet, and is the main cause of the present epidemic, whilst the absence of variety and deficient quantity of food hastened the development of scurvy."

Bellingham summarises the results of observations in Ireland as follows: "That this disease did not make its appearance until after the people had been deprived of their accustomed diet for several months, that the disease prevails only among that class of the population whose diet consisted formerly, in a great measure, of the potato, and who, as long as they had that vegetable in abundance, enjoyed a perfect immunity from it; the subjects of the preceding cases appear all to have had a sufficiency of bread, others had meat in addition, with sometimes wine or porter; none suffered from an absolute deficiency of food, but all agreed in not having used fresh vegetables from the period of the failure of the potato crop of last year." "In four fifths of the cases reported to me," says Curran, for Dublin, "bread and tea or coffee, was what the patients had been living on when attacked; the others had been using grains of various kinds, or grains and flesh or fish, but in no single instance could I discover that green vegetables or potatoes, had formed a part of their regular dietary."

¹ L. c., p. 427.

Sibbald, who studied the epidemic in the Kent County Asylum, says that as soon as potatoes were procured, "although at an exorbitant price," and the ailing and healthy began to have them at meals, the former soon got well and no new cases occurred among the latter. The same conclusion was come to by Ritchie, Shapter, Anderson, Christison, and others. It is the opinion of nearly all the authorities¹ on the epidemic of scurvy in Paris during the siege by the German troops in 1870-71, that the cause of the malady was neither overcrowding (in such places as prisons, barracks or military hospitals), nor cold and wet, nor the use of salt meat; neither was it scarcity of food in general that was to blame; but it was when the supply of fresh vegetables, and particularly of potatoes failed, that the disease broke out.

In nearly all the epidemics of recent years among the British and native troops in India—at Rangoon in 1824, Nusserabad in 1834, Cannanore in 1832, and during the siege of Lucknow in 1857—the cause of the sickness could be traced to the want of fresh vegetables, either from failure of the crop or the difficulty of introducing supplies. "Little or no fruit has been procurable in Rangoon," we read in one of the accounts,² "and the only vegetable available to the troops has been wild spinach or country greens, and that even in a very limited quantity."

The real cause of the epidemic at Cannanore in 1832 is stated by Henderson to have been "the scarcity and high price of good vegetables;" and of the epidemic in Lucknow, Greenhow says: "Considering the total want of vegetables and the absence also of lemon-juice and vinegar, the wonder is that more persons did not suffer in this way. Rice was served out latterly, of course, in very reduced quantity; but rice cannot be considered a good substitute for good vegetables, a fact proved indeed by the experience of this siege. The best treatment for the complaint, and an evidence too, if such were needed, of its real origin, was the portating again of fresh vegetables. When the supply of these was opened up, scorbutic disease began to disappear."

Under the same circumstances scurvy appeared in 1862 in the Army of the Potomac in the American Civil War. There also fresh vegetables failed altogether, and it was only with the greatest difficulty that a small quantity of potatoes and cabbage could be procured, "the beneficial effects of which were marvellous," as Herr says in his report. The same consequences of the want of fresh vegetables, and the same curative effects of them in scurvy when they were procurable, had often been experienced before by the surgeons of the United States Army at remote posts in the Western States, and in Texas and New Mexico. Thus Madison, speaking of the scurvy at Fort Randall, Dakota, in 1857, says: "During the last winter the whole command was more or less affected before we received the Irish potatoes, which had left St. Louis

¹ Delpech, Georgesco, Bucquoy, Charpentier, Lusègue, &c.

² 'Madras Quart. Med. Journ.,' 1839, i, 209.

in the fall and had to be deposited one hundred miles below, and afterwards hauled up, frozen, in waggons. They did not reach us before the first week in January. As soon as a liberal issue was fairly commenced, and the men compelled to eat them raw as well as cooked, their convalescence was most rapid and recovery complete. After the potatoes gave out, the supply of which was not abundant, the disease showed a strong disposition to return."

Johns,¹ for Fort Laramie, Nebraska, and various other² authorities, have come to the same conclusion. Again, Logan points out that scurvy appeared among the gold-diggers in California when they were entirely without fresh vegetables or the corresponding vegetable juices, having to live on flour and half-putrid meat which had become hard in the pickling and drying. The epidemics of scurvy that have been observed time after time in Russia have been shown to depend on the etiological factors of which we are speaking; and in the Crimea, according to Heinrich, in Jekatermoslav, according to Sachs,³ and in other of the southern governments, the endemic prevalence of the malady has been essentially connected with the want of fesh vegetables in the winter and spring seasons. From Finmark we have it stated by Walter that scurvy is rarely seen among the Finnish and Lapp population of the country, who collect large quantities of sauerkraut (*Rumex acetosa*) during the autumn and eat it with milk in the winter; whereas the malady is very common among the Quaens and Northmen who do not follow the same practice.

In Sweden, says Dalberg (l. c.), the disease used to be prevalent in those districts where the inhabitants suffered from the want of fresh vegetables. Under those circumstances, also, it has occurred time after time in Iceland, according to Holland,⁴ Schleisner⁵ and Hjaltelin, and, according to Sexby,⁶ in the Shetland Islands, especially in seasons when the potato crop was a failure.

The unusually frequent, and even endemic, occurrence of scurvy in Rajpootana, Malwa, and other parts of north-western Hindostan, is explained, as Moore and Lucas think, by the fact that these tracts of country, which are very barren of every kind of growth, produce no anti-scorbutic plants; but the malady breaks out all the sooner and spreads the farther, the greater the want of vegetable food in general. From information furnished by Pechey, scurvy is not uncommon among the shepherds in the interior of Australia, who live mostly on bread and salt meat. Whenever there is a fall of warm rain, many juicy plants at once shoot up, which, although not agreeable to the taste,

¹ 'Statist. Report on the Sickness and Mortality in the Army of the U. S., 1859-60,' Washington, 1860, 45.

² See Forry, for Council Bluffs (Iowa); Perin, for Fort McIntosh (Texas); Bartholow, for Fort Bridger (Wyoming); McBride, for Camp Bull, ll. cc.

³ 'Med. Ztg. Russl.,' 1848, 37.

⁴ 'Edinb. Med. and Surg. Journ.,' 1812, April, 202.

⁵ 'Island undersögt, &c.,' 48.

⁶ In Dobell's 'Reports.'

are nutritious; and they would appear, from the language which the writer uses, to have a good effect as antiscorbutics. Unfortunately, rain seldom falls in any quantity, and the supply of these plants is never more than a scanty one.¹

There are extremely plentiful materials for forming an opinion on the question before us, to be found in the experience acquired in prisons and on board ships as to the influence of a want of fresh vegetable food on the production of scurvy. Classical instances of the first kind are the epidemic of 1840 in the prison of Clairvaux, where potatoes were withheld from the prisoners, and herrings supplied instead; also the observations of 1823 and 1840 in Millbank Penitentiary, where it was only the prisoners sentenced by court martial, whose diet contained hardly any vegetables, and in particular the very smallest allowance of potatoes, that took scurvy, the other inmates of the prison, who had a better diet, remaining well. Baly, who reports these facts, adds that scurvy had occurred time after time in many other English prisons, where "potatoes or green vegetables were given only occasionally on Sundays, when the prison garden would furnish them." Scurvy broke out also, under these or similar circumstances, in the military prison of Alessandria in 1840, and in the prison of Christiania in the years 1844-47.

Regarding the occurrence of scurvy in Swedish prisons from 1848 to 1877, Heyman gives the following interesting details:—In the first ten years (1848—1857) the number of cases of scurvy was 52·5 per 1000 prisoners; in the second ten years (1858—1867) it was only 32·9; and in the third period (1868—1877) it fell to 17·0. From 1848 to 1877 there had been two diets in force; the one (down to 1862) contained succulent

¹ This leads me to recur to the above-mentioned (p. 545) account of Beckler on the outbreak of scurvy among the members of Burke's exploring party, an outbreak, which was caused, according to Beckler, by the drinking of bad water. If we examine more closely the list of things provided for the expedition, we find that besides twenty gallons of lime-juice, which, as we learn from Beckler, was left behind before they came to the Darling river, the only antiscorbutic food was preserved vegetables, as to which our authority himself writes: "Of our preserved vegetables there is little good to be said. We had only a few packages of dried assorted vegetables, the properties of which had been so preserved by the mode of preparation that we were justified in expecting them to make up in a measure for the want of fresh vegetables. By far the most of our supply consisted of unassorted vegetables of various kinds, dried and pressed, which I am bound to say, without bias, that I consider to have been useless." Taking this into consideration, along with the fact that the explorers had to endure great fatigue, it becomes far from improbable that the bad water was only a predisposing cause, although it may have been a potent one, in the outbreak of the malady. There may be another explanation of the fact that the relief-expedition which was sent out, and which took the same provisions with them, escaped the disease; they began their journey after the rains, and they would not only find good water, but would also be able to use with their food those juicy plants mentioned by Pechey. However, there is nothing said on that point in Beckler's account.

vegetables for each prisoner in the proportion per week of a pint and a half of potatoes, two pints and a half of carrots, &c., and a pint of sauerkraut; the other (after 1862) contained three pints more of potatoes. But those vegetables were supplied only in winter, the equivalent diet in summer being bread, flour, barley and peas. Now, the remarkable thing is that nearly all the epidemics in the Swedish prisons began in summer and died out towards the end of the year. Further, since 1865, the prisoners have had leave to buy food with the wages that they earned; and during the summer months they have made use of this permission chiefly to buy potatoes. Lastly, the most efficient means of combating an epidemic has been found to be the addition of potatoes to the rations; and of late years that has always been done the moment any indications of scurvy showed themselves. According to Porter, epidemics of scurvy in Indian (Madras) gaols mostly occur at the time of the monsoon (July to September), that is to say, at the time of the year of all others, "when the months preceding are most likely to have had a scanty supply of fresh vegetables."

Of the production of scurvy on board ships in consequence of a deficient supply, or no supply at all, of fresh vegetables for the crew, there were two striking instances as early as the first half of last century, in Admiral Hosier's disastrous expedition¹ with the English fleet against Porto Bello, in 1726, and in Lord Anson's expedition to the Pacific in 1741. In the latter part of that century we have the hardly less instructive instance narrated by Curtis, of the outbreak on board the fleet during a voyage from England to India in 1781; the fresh vegetables taken on board at St. Jago (Cape Verd Islands) having been exhausted after a voyage of several months, the men refused to eat the pickled cabbage that remained, and scurvy broke out among them; but it vanished a few days after, when the sick were landed on Johanna Island (in the Mozambique Channel) and supplied with fruit and vegetables. Hardy² says of the outbreak of scurvy in 1838 on board the *Palinurus* frigate cruising on the East Coast of Africa, that "none of the obvious causes of scurvy were present except the want of fresh vegetables, for the ship's company had abundance of good dry food, fresh meat and good water." The U. S. frigate *Columbia* having been cruising for several months in tropical waters, had an outbreak of scurvy on board after the meat went wrong and the fresh vegetables ran short; but the sickness very soon ceased when the vessel arrived at a Chinese port and the crew were supplied with fresh vegetables. Of twenty-eight officers, only three took scurvy, and these had shared the diet of the crew.³

¹ ["Sent in this foul clime to languish,
Think what thousands fell in vain,
Wasted with disease and anguish,
Not in glorious battle slain."

Ballad of Admiral Hosier's Ghost.]

² 'Transact. of the Bombay Med. Soc.,' 1839, ii, 256.

³ Coale, 'Amer. Journ. of Med. Sc.,' 1842, Jan.

A very interesting contribution to the matter under debate, is given by Foltz in his account of the outbreak of scurvy in the U.S. blockading flotilla in the Gulf of Mexico, in 1846. To show the influence of a deficient vegetable diet on the production of scurvy, he adduces the fact that he had often seen the disease on board whalers; and whenever he had seen it, it had always been after the best antiscorbutic elements of their food, namely, fresh potatoes, had been used up, while, on the other hand, the malady never appeared so long as these held out. This is confirmed by a statement in Morgan's¹ account of the scurvy on board the troop-ship *Lismoyne*, on a voyage to India; among the recruits which she carried those only took the disease who did not eat their ration of potatoes but exchanged it with their messmates for salt beef. Lilienfeld's² experience of an outbreak of scurvy on board a Dutch man-of-war, during a voyage round the world in 1849, leads him to the conclusion that "the deprivation of fresh vegetable food is the principal cause of scurvy in ships."

Even in recent times the ships of the French navy have several times suffered severely from scurvy owing to the want of fresh vegetables. Of special interest is the account by Léon, of the epidemic on board the transport *Castiglione*, which was one of the vessels employed in conveying the French troops back from Mexico, in 1867. The first symptoms of the epidemic appeared as early as the twentieth day out from Vera Cruz, and in the course of the next ten days it had extended so much that, of the whole crew (who were the only class on board to suffer), one third were more or less ill. Notwithstanding that fresh meat, acidulated drinks, and lime-juice were served out, the epidemic did not come to an end; and it became necessary to make for the Azores, where large quantities of fresh vegetables, potatoes, cabbages, and the like were taken in. From the day when these were served out to the healthy and the sick, new cases ceased to occur among the former, and convalescence proceeded so rapidly among the latter that, when the ship arrived at Toulon a fortnight after, all the more considerable signs of illness had disappeared. Léon points out that neither the weather nor the hardships of the service, nor any depression of the feelings had occasioned the epidemic, but solely the want of fresh vegetable food, which was not to be got at Vera Cruz. It is worthy of note, at the same time, that cases occurred only among the crew, who had been already exposed in part to the same deficient diet during the voyage out from France to Vera Cruz; and that not a single case of scurvy occurred in any of the other ships of war or transports, none of which sailed direct to France from the Mexican coast, but touched at the West Indies, where they received abundant supplies of fresh vegetables.

Again the disease has several times broken out under the same influences on board French ships of war carrying convicts from France to New Caledonia, as, for example, in 1866 on board the frigate *Sybilie*,³ 1867 in the frigate *Iphigenie*,⁴ and 1873 in the transport *Orne*.⁵ Ayme⁶

¹ L. c., p. 25.

² 'Med. Times and Gaz.,' 1854, Dec., p. 586.

³ Normand.

⁴ Caurant.

⁵ Ayme.

⁶ L. c., pp. 26, 31.

says : “ La privation d'aliments végétaux (frais), est la seule cause puissante, décisive, que nous puissions invoquer pour explicier l'épidémie de l'Orne . . . toutes les autres conditions restant les mêmes après notre relâche à Melbourne, la présence de vivres et de végétaux frais dans la ration de l'équipage et des déportés, a suffi pour arrêter, d'une manière brusque, les progrès de la maladie.”

Lastly, it should be stated that scurvy has not unfrequently appeared in vessels of the mercantile marine and in whalers, just as in ships of war, when their crews have been without fresh vegetables for a considerable time. Under the same circumstances, several of the Arctic expeditions have suffered from scurvy; for example, the crew of the *Investigator* in the spring of 1852, when the ship had been out, as Armstrong tells us, two years and a quarter and the rations of fresh vegetables, potatoes in particular, and of lime-juice had been reduced considerably; and, again, in 1875 in the *Alert* and *Discovery*, the cause of the outbreak in their case being assigned, upon evidence given by experts at an official inquiry, to not using lime-juice as a substitute for fresh vegetables.

In consideration of these and many other observations of the same tenour, it is impossible with any show of reason to deny the importance of a scarcity or absolute want of fresh vegetables in the diet as the real cause of scurvy; and we should perhaps not err if we were to find in that view of the causation the explanation of a fact which, it seems, is not to be denied, namely, that scurvy was not only of more frequent occurrence in former centuries than it has been of recent years, but also more widely spread, especially in the higher latitudes. Besides the enormous extension and multiplication of the means of transit, which makes it much easier than formerly to compensate for the failure of fresh vegetables in regions remote from traffic, there is, first and foremost, the benefit that has accrued to the peoples of the Eastern Hemisphere during the last two centuries in the cultivation of the potato, the most effective anti-scorbutic in the dietary. There is also the more systematic and more general cultivation of all kinds of vegetables, an industry which even in the sixteenth century was in so backward a state in Northern Europe that Catharine of Arragon, queen of Henry VIII, desiring to have a salad for the table, had to send her gardener to the Low Countries to procure it.

Garrod was led by the consideration that scurvy came to so great a height in the United Kingdom during the potato famine of 1846 and 1847,

to analyse the most usual articles of diet so as to ascertain their proportion of carbonate of potash, a substance in which potatoes are unusually rich. He arrived at the results in the following table, which shows the number of grains of carbonate of potash in an ounce of each substance :

	Grains per ounce.		Grains per ounce.
Large potatoes (boiled)	1·875	Pease	0·529
Small „ (raw)	1·310	Beef (salted)	0·394
Lime-juice	0·852	Onions	0·333
Lemon-juice	0·846	Wheaten bread	0·258
Unripe oranges	0·675	Cheese (Dutch)	0·230
Mutton (cooked)	0·673	Flour (best)	0·100
Beef (raw)	0·599	Oatmeal	0·054
Salt beef (slightly salted).	0·572	Rice	0·010

It follows from this table that all those articles of diet which have been especially apt to develop scurvy when used exclusively, contain carbonate of potash in smaller quantity, sometimes in much smaller quantity, than those which are never followed by scurvy and have been proved to be the best antiscorbutics (potatoes and lime-juice). Having regard at the same time to the fact (not altogether certain, however) that the blood in scurvy is characterised by a deficiency of potash salts, Garrod draws the conclusion that the cause of the malady is to be attributed to a diet poor in carbonate of potash.

§ 195. SCURVY UNDER EXCEPTIONAL CIRCUMSTANCES.

Highly as we must always rate this etiological factor in the production of scurvy, it is at the same time not to be denied that the malady has developed now and again under circumstances which make it at least improbable that the cause had been an error in diet, or more particularly the want of fresh vegetables. It is true, indeed, that the observations adduced in proof of this are not all of equal value, and we shall have to reserve our opinions as to their trustworthiness. On board French and English ships-of-war, such as the frigate on the voyage from L'Orient to China mentioned by Lagarde, and the ship of the English navy bound for Calcutta quoted by Wrench,¹ outbreaks of scurvy have occurred although the crews were supplied with lime-juice and preserved vegetables; and although these observers draw the conclusion that the disease may originate indepen-

¹ 'Med. Times and Gaz.,' 1867, March, p. 317.

dently of the want of fresh vegetables, yet it should be kept in mind that lime-juice is perhaps not absolutely trustworthy in its prophylactic action, that it is often adulterated,² that it spoils easily, especially in the tropics, that it is not unfrequently administered in too small doses, and that preserved vegetables are by no means a substitute for fresh vegetables. Other instances which have been adduced to show that there was no cause of scurvy discoverable in the dietary of those who took it, such as the case of the lunatic asylum at Moorsshedabad (Bengal) mentioned by Burt, and Maupin's cases in Algiers, want the force of conviction, inasmuch as it does not appear from the evidence what these observers understand by a "suitable" diet. In still other papers written with the object of proving that scurvy has developed apart from defects of diet, the want of clearness and of critical acumen on the part of the observers is conspicuous.

Thus Le Vicaire traces the epidemic of scurvy among the French blockading squadron in the Mediterranean in 1827-29 to the amount of tobacco-smoking among the crews, although he tells us besides, that they lived upon biscuit, salt meat, and dried vegetables. In his account of the scurvy in 1872 on board the steam transport *Var* carrying convicts to New Caledonia, Ledrain lays the stress upon the confinement of the convicts in damp, dark, and ill-ventilated berths; but he adds, "quant aux végétaux frais nous ne pouvions en prendre qu'une faible quantité, la place manquant pour les loger." The cause of the epidemic of scurvy in the garrison hospital of Givet in 1846 was attributed by Scoutetten to the low and damp situation of the building; but he mentions also that the food of the troops was insufficient, they having been put on short allowance to the extent of one-third or one-half.³ Seeland also, who is inclined to see "miasma" in so many things, denies the influence of diet upon the production of scurvy in the regions of the Amoor; for, as he assures us, the store of greens usually

¹ "The lime-juice of commerce," says Beckler (l. c., p. 239), "is one of those articles adulterated in so many ways, that we can hardly depend upon it."

² On Moreau asking the reason for this lowering of the rations, Scoutetten stated that he was not able to speak out on the matter: "Je serais obligé pour y répondre," he adds, "de produire des chiffres qu'il ne m'appartient pas de faire connaître ici."

holds out through the winter and spring—he does not say what happens when it does not hold out—and it is only now and then during the latter half of winter and during the spring that the potatoes fail from going wrong in the damp cellars.

There is one other point that I must call attention to, which does not seem to me to have been estimated at its full importance in considering the influence of diet, and vegetable diet especially, in the problem of scurvy. I mean the predisposition to, or immunity from the malady, howsoever brought about, among some people as compared with others living under the same circumstances. How do we explain the fact that, *cæteris paribus*, one group becomes subject to scurvy, while others remain free from it? In the epidemic among the British troops in Cape Colony at the time of the Kaffir War of 1836, there was only one regiment that really suffered, although all the regiments were equally in want of vegetable food. The explanation is, as Morgan has pointed out, that that regiment had to undergo particularly great fatigue in making long marches during very bad weather, and it was the one, therefore, in which the deficient diet made its effects most felt. In the slight epidemic of 1873 among the garrison of Prague, according to Kirchberger, it was only the troops from Bohemia that suffered, the Galizian regiments remaining exempt although they had the same rations. The explanation may be found, as that writer suggests, in the fact that the Bohemians are on the average much better fed at home than when on service, whereas the contrary is the case with the Galizians. We are not informed, however, of the extent to which this lowering of the rations relatively to the usual allowance of food, affected the actual quantity of fresh vegetables in the diet. But this factor of a predisposition to scurvy, or immunity from it, acquired by being habituated to a particular diet, appears to me to come out very decidedly in the circumstances under which the disease attacks people in high latitudes, as in Kamschatka,¹ Greenland,² and Hudson's Bay Territory.³ In these latitudes scurvy occurs solely among strangers, whenever they suffer the want of the fresh vegetables to

¹ Bogorodsky.

² Lange.

³ Smellie.

which they have been accustomed ; while the natives, who have been accustomed from their birth to live principally, if not altogether on animal food, bread and the like, remain quite free from the disease.

But besides these observations, which are still capable of being interpreted in favour of the theory that the malady is caused by the want of fresh vegetables, we have a series of epidemics of scurvy, the origin of which, as credibly alleged by those who observed them, cannot be referred to want of fresh vegetables, or to erroneous or deficient diet at all. Such are the outbreaks narrated by Dicenta and Cless for the prison of Ludwigsburg from 1852 to 1858, by Opitz among the Austrian garrison of Rastatt in the winter of 1851-52, by Döring among the French prisoners of war at Ingolstadt in 1871, by Kühn at the penitentiary of Moringen (magistracy of Hildesheim) in the winter of 1875-76, and several of the epidemics on board ships of the English, French and German navies. Such instances, rare though they be, serve to show that the disorder of nutrition which underlies the scurvy, may as an exceptional thing originate under the influence of other debilitating factors, mostly associated with a life spent in prisons, barracks or ships. But the rarity of these cases is, to my thinking, a proof that all those commonplace injurious influences, such as damp soil, cold and wet weather, bad air from overcrowding and want of ventilation, and bad drinking-water, which are incessantly in operation without any epidemic of scurvy breaking out, are not to be reckoned as real factors in the pathogenesis. In the rare instances referred to, we have rather to assume some peculiar modification of hygienic defects of that kind, or perhaps a certain *ensemble* of them, concerning the nature of which the published records tell us nothing ; and that is an assumption which we might make without going so far as to postulate a *specific virus of scurvy*.

§ 196. ATTEMPTS TO PROVE A MIASMATIC, INFECTIVE OR
CONTAGIOUS ORIGIN FOR SCURVY.

There has certainly been no lack of hypotheses of a miasmatic cause for scurvy. In the last century the doctrine was maintained by Poissonier-Desperrières,¹ and others, that scurvy was allied in its causation to the malarial diseases; and in these days of the malaria-craze there have been many attempts made to construct some such hypothesis as would secure for scurvy a place among the infections, or even among the communicable and contagious diseases. Besides Scoutetten and Dévé, who are also in favour of the malarial nature of the malady (Dévé would have it that malaria occurs on board ship), we have had within the last twenty years such writers as Krüggula, Villemin, Kühn, Petrone, and Seeland pronouncing in favour of the *miasmatic or infective character of scurvy*. The noxious influences which have hitherto passed current as the cause of the malady do not fulfil, says Krüggula, the requirements of real causes; scurvy is chiefly found in localities which are known to be the breeding-places of the acute infective diseases, and it frequently shows itself in company with the latter; it has often happened that scurvy has broken out as the immediate effect of substances directly involved in the processes of decomposition, such as stinking fish, foul water, and the like; and in the majority of cases its production can be explained much more naturally by infection than by any of the hypotheses hitherto current. In its origin scurvy is associated with typhus, and in respect of its being non-contagious it connects with intermittent fever. (Dr. Krüggula does not appear to be familiar with the large body of facts which furnish, I may almost say, a mathematical proof of the origin of the disease from those errors of diet which I have entered into in detail; and that is perhaps the reason why he finds his explanation of the disease to be a more natural one than that of other inquirers, whose theory rests not upon assertions but on observations, and who have accordingly set up no mere hypothesis, as he has done.) Villemin also,

¹ 'Traité des maladies des gens de mer,' Par., 1767.

with a veritable profusion of arbitrary and erroneous assumptions, seeks to prove that scurvy is allied in its causation to the typhous diseases, and, in contrast to Krüskula, he claims contagious properties for it as well. Like typhus, he says, scurvy has primary foci on the shores of the German Ocean, the Baltic, and the Black Sea; and from these it is carried abroad by the shipping. With this prodigy of a theory, he couples the statement that epidemics of scurvy very often run their course parallel with epidemics of typhus, and that the best way to obviate the spread of the disease is removal to a distance from the morbid centre. This doctrine of Villemin has met with a truly brilliant refutation at the hands of Le Roy de Méricourt, whose ample experience and sound literary training have enabled him to exhibit the reasons advanced by Villemin as untenable, and whose unhesitating conclusion is that a deficient diet, and especially the want of fresh vegetables, is the essential factor in the causation of scurvy. Shortly after, it was pointed out by Galliot also, that the epidemic of scurvy which sprang up on board a French ship-of-war on the voyage back from Iceland was not due to a "miasma scorbuticum," but to the want of fresh vegetables, and that the disease ceased abruptly as soon as fresh provisions were procured.

The hypothesis of the miasmatic nature of scurvy has been expounded after another fashion by Kühn, with greater skill perhaps but, in my opinion, with just as little success. The notion of "scurvy" is enlarged by him, so as to include the macular disease of Werlhof, which is a totally different thing from scurvy, as well as "stomacace," which has nothing else in common with scurvy except that it is an affection of the gums. Next, he distinguishes between a scurvy of inanition, as he calls it (or one that is induced by errors of diet), and an infective scurvy, which is nearly related to the former in symptoms but is quite different in its etiology, being caused by a miasma; and to the latter he ascribes contagious properties, just as Villemin does. If we examine more closely his type of infective scurvy, filling in details from his clinical histories, it is impossible to escape the impression that he has widened the idea of "scurvy" to an inordinate extent, taking as signs of a scorbutic affection slight reddening or

swelling of the gums, even in persons with decayed teeth. On that wide basis he makes his diagnosis of scurvy; and then he creates a number of forms of scurvy, such as scorbutic peritonitis, bronchitis, angina, pneumonia, and rheumatism (reminding one of Sydenham's "febris dysenterica sine dysenteria," or his "febris variolosa sine variolis"), for which he assumes a miasmatic infection, probably by the agency of putrefactive bacteria, inasmuch as he would connect the origin of the malady with the fact of the patients living in overcrowded and unventilated places. Finally, Seeland has gone one step farther—he has seen the bacteria. According to him the cause of scurvy along the shores of the Amoor is a fungus, which grows luxuriantly in the houses of the better-off class of officials, owing to the dampness of the soil; while the natives, whose huts are made of hard-baked clay, are exempt from the malady.

But the doctrine of the *contagious nature of scurvy*, asserted by Echthius, Horst, Poupart, Trotter, and other writers of the eighteenth century, and lately revived by Villemin, Kühn, Murri, and Cantu, is quite untenable, inasmuch as it goes against all historical and clinical experience. Villemin's assertion that the disease is indigenous on the coasts of the German Ocean, Baltic and Black Sea, whence it has spread by maritime commerce after the manner of a contagious disease, such as typhus, is so fanciful that it does not require to be seriously met; and the other evidence which he brings forward in support of his view, rests, as Le Roy de Méricourt has shown, upon an erroneous reading and stating of the facts. There is just as little reason to find any evidence of the communicability of the disease in Kühn's observations of scurvy occurring in those who had come into close contact with scorbutic patients, the less so that the scurvy in the cases in question was of the ambiguous kind. Inspired by Kühn's writings, Murri made experiments on four rabbits to produce infection by means of blood drawn from the vein of a scorbutic patient. After subcutaneous injection of the blood, small hæmorrhagic spots appeared on the ears of the animals, febrile symptoms (rise of temperature) having sometimes preceded them; and the anatomical examination revealed slight effusion of blood on the dura mater and

pleura, and in one of the rabbits on the peritoneum also, and in the liver and spleen. Murri does not go so far as to conclude that these phenomena have any significance in the way of proving the communicability of scurvy. Cantu also, who has repeated Murri's experiment on two rabbits, and with the same result, is equally cautious, contenting himself with verifying the facts. That these animals really acquired a scorbutic disease follows neither from the account of the symptoms nor from the conditions found *post mortem*.

Summing up briefly the facts and arguments of this chapter, I am led to conclude, as regards the genesis of scurvy, that the disease is most of all associated with want of fresh vegetables in the diet, perhaps with the insufficient supply of salts of potash (combinations with the vegetable acids); that it breaks out the more promptly and the more severely, the greater the antecedent action of other debilitating things on the organism, predisposing it to sickness; that there are certain other errors of hygiene, for the present not to be more accurately particularised, which induce the disorders of nutrition that underlie scurvy, although they do so much more rarely than the first mentioned; but that there is no warrant to speak of a miasmatic or infective origin of the disease, while a contagious property is to be denied of scurvy in the most absolute terms.

The Ponos (or "Pain") of the Islands of Spezza and Hydra.

§ 197. CLINICAL CHARACTERS AND MORBID ANATOMY OF
"PONOS"—A CONSTITUTIONAL DISEASE OF INFANCY.

In connexion with scurvy I have to speak of a peculiar disease affecting young children only, which is endemic in the Greek islands of Spezza and Hydra on the eastern side of the Peloponese, off the coast of Argolis. It is only recently that the attention of the profession in Greece has been drawn to this malady, which is known by the hardly distinctive name of Ponos or "pain." It is undoubtedly to be viewed

as a constitutional affection deeply rooted in errors of nutrition; but the clinical and pathological studies hitherto made of it have not yielded any definite information as to its nature.¹

Clinical history.—The onset and course of the disease are always with fever, which has an intermittent or irregular type and exhibits many fluctuations from first to last, assuming the character of hectic when the issue is unfavorable, as it usually is. The commencement of the malady is indicated by some change in the child's strength and spirits; it loses its sprightliness, betrays feebleness in its gait, becomes sad, and disinclined to play. The skin acquires a pale or straw-coloured tint; and progressive emaciation becomes noticeable, although the appetite keeps up for the most part, not unfrequently indeed becoming ravenous, with a peculiar craving for highly seasoned dishes and for spirituous drinks. Meanwhile the belly enlarges owing to the gradual swelling of the spleen, which often attains so enormous a size as to reach the middle line of the abdomen. In many cases the spleen is painful or tender to the touch, whence the name given to the disease; but it oftener happens that the tenderness is wanting, notwithstanding that the organ is much enlarged. As an exceptional thing, there may be slight enlargement of the liver; and swelling of the lymphatic glands in the neck has been noticed in a few rare cases. The digestion is always out of order, and it is not uncommon for vomiting to come on after food. At the outset of the disease there is usually constipation, but later on the dejecta assume the character of diarrhœal or dysenteric. A very characteristic sign is the penetrating odour of the urine, which becomes noticeable from the very beginning of the illness and continues all through it; usually there is a sediment in it (possibly urates), but rarely or never albumen. One of the most frequent symptoms throughout the disease is bronchial catarrh.

At a later stage a variety of exudative phenomena occur—the profuse intestinal discharges already mentioned, dropsy (œdema at first and then ascites), and colliquative sweating; thereafter, hæmorrhages in the form of petechiæ or extensive ecchymoses of the skin, bleeding from the nose and from the bowel, and above all bleeding from the gums. The latter at length show the signs of a scorbutic affection; an ichorous discharge comes from them, the teeth become loose and drop out in the end, and in some cases even necrosis of the jaws has been observed. These are the incidents, along with general marasmus, that usher in the fatal result, if it have not been now and then anticipated by intercurrent local disease, such as broncho-pneumonia, meningitis, or peritonitis.

¹ The account that follows in the text is based upon the papers of Karamitsas ('Gaz. des hôpit.,' 1880, No. 19, p. 147), and Stephanos ('Gaz. heb. de méd.,' 1881, Nos. 47 and 51, pp. 750, 813). These papers make reference to the notices of the malady which have appeared since 1835 in the medical journal, 'Γαλῆνος,' published at Athens.

The illness has usually a fatal termination; it is only in exceptional cases, when a suitable diet and tonic remedies (quinine, iron, and iodide of iron) are employed that a cure results, and then only if the disease have been treated in its early stages. In the case of very young children the best hope, although that is not much, lies in the choice of a good nurse. There are many remissions and exacerbations in the course of the malady, which usually lasts from one to two years. Sometimes it runs its course under very intense symptoms, and ends fatally in two or three months.

Morbid anatomy.—Only one *post-mortem* examination has been made hitherto, of which an account is given by Stephanos. There were no traces of leukæmic or pseudo-leukæmic, or of tubercular or malarial lesions. The capsule of the spleen was very firm, and the trabeculæ a good deal thickened; the substance of the organ was swollen, but there was no actual pigmentation and no excess of colourless corpuscles. In the right lung there was an abscess reaching to the pleural covering; there was also commencing cirrhosis of the liver (the child had received alcoholic drinks during its illness); the kidneys were congested, and the bronchial and mesenteric glands normal, although some of them were slightly enlarged.

This disease occurs, as we have said, only during the first years of childhood, usually covering the period between the first and second dentition; in most cases it begins to develop as early as the eruption of the first incisors. Only in very rare instances are children attacked at the age of four or upwards. It is said (by Stephanos) to be rather more common in boys than in girls.

§ 198. HISTORY OF "PONOS."

Our information about the "ponos" goes no farther back than 1835, in which year Röser directed attention, in an address before the Medical Society of Athens, to enlargements of the spleen in young children on the island of Spezza, the cause of which he traced to the use of water from cisterns. Reference was afterwards made to it by Pallas,¹ who pointed out the hæmorrhagic phenomena and their resemblance to those of scurvy. It was not until 1871 that this malady received general recognition at the hands of the profession in Greece.

The disease, as we learn from the most recent inquiries, is endemic nowhere but in the islands of *Spezza* and *Hydra*; in other parts of Greece it has never been seen hitherto.

¹ 'Annali univ. di med.,' 1842, c. ii, 61

It used to be commoner formerly than it is now ; even at the date of Pallas's article, there were few families in Spezza but had lost one child at least by it. There have, indeed, been considerable fluctuations in the number of cases from time to time : according to the latest accounts, there were twenty to twenty-five children with "ponos" in Spezza out of a population of 7500, and ten to fifteen in Hydra out of a population of 7300.

§ 199. CIRCUMSTANCES UNDER WHICH "PONOS" OCCURS.

Climate and *season* have no influence on the production of the disease. Neither has the kind of *soil* ; for it occurs equally on high and low ground, on rocky and damp soil, on the chalk soil of Hydra and on the volcanic (gompfolithic) formation of Spezza. *Social rank* also would appear to be of no real significance ; the cases of it are uniformly distributed through all classes from the rich downwards, and in all kinds of dwellings, from the larger sort of well-kept and well-aired residences to the hovels of the poor. Neither is there any real causative element to be discovered in the *food*, which differs in no respect from that used in other parts of Greece where the disease is unknown. It follows also that the much-blamed *use of rain-water* is not the cause, for the malady has been found in families who take their water-supply from deep wells. The view adopted by Jeanakopulos, that it arises from *malarial infection*, is quite untenable ; for on neither of the islands is there any malaria at all, and the disease is one that affects a time of life when malaria is least apt to be felt. Moreover, it is limited to certain families.

It is this last-mentioned fact, together with the fact that the parents of children with "ponos" had suffered from serious diseases, especially pulmonary consumption, that so far bears out the opinion generally current among the inhabitants of Spezza, that the question is one of *hereditary conditions* or of a congenital predisposition. We are completely in the dark as to the actual disease-producing agent ; and Stephanos, in view of the fact that the malady cannot be

brought into causal connexion with any of the influences before mentioned, throws out the suggestion whether infection or *parasitism* may not be at the bottom of it! Lastly, it should be said that the opinion held by Karamitsas of the disease being a kind of splenic leukæmia, does not find the smallest support either in phenomena observed during life, or in the post-mortem appearances, or in the examination of the blood (that author himself found the coloured corpuscles considerably fewer and only some traces of leucocytes left); and therefore Stephanos thinks himself warranted in decidedly rejecting the idea of an affinity between the disease and leukæmia or pseudo-leukæmia.

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CHAPTER XVI.

BERIBERI.

§ 200. CLINICAL CHARACTERS. THE PARALYTIC AND THE DROPSICAL FORM.

The malady known under the name of "beriberi" or other colloquial designation,¹ which is endemic in many tropical and sub-tropical parts of Eastern Asia, was till not very long ago a riddle in its pathology and causation. And although the latest observations of English, German, Dutch, and Brazilian practitioners have thrown much light upon it, we are still far from being able to assign to it a definite and undisputed place in the nosological system, to characterise its nature with precision, and least of all to come to any positive conclusion as to its genesis or the causes that under-

¹ The etymology of the word "beriberi" has not been explained. Its origin is certainly not Arabic, but Hindustani or Malay. In the Malay language "biribi" means an "abrupt and tripping gait" (*Platteenu, 'Geneesk. Tijdschr. voor Nederl. Indie,'* x, 665). According to Bontius, who was familiar with the disease in Java, the word is derived from "bharyee," meaning "sheep." Marshall, on the other hand, derives it from the Cingalese word "bhargee," or "feeble gait," and Herklots from the Hindustani word "bharbari," meaning "swelling," *i.e.* œdematous swelling. In Banka the disease is called "binas" or "apooi," as well as "pantjakit niloe" or "siloe;" in Java "loempoe;" on the coast of New Guinea "pantjakit papoea." In Japan it is known under the name of "kak-ké," a term of Chinese origin, derived from the words "kiaku," meaning "leg," and "ka" or "ki," meaning "disease," and equivalent therefore to "disease of the legs" (Scheube). In the French Antilles it is called "maladie des sucreries;" in Cuba, "hinchazon" (dropsy) "de los negros;" in the Brazilian province of Matto-Grosso, "perneiras" (disease of the feet); and in Minas Geraes "inchacão" (œdema). The term "barbiers" is undoubtedly a French corruption of "beriberi." Vinson (I) however, who was born in Réunion, and became familiar with the disease, says that it is another disease which is denoted by "barbiers;" but it would appear from the description of that other malady, that it is itself the acute pernicious form of beriberi.

lie it. The recently published works of Wernich¹ and Scheube on the disease in Japan, where it is known as "kak-ke" have made the profession in Germany more intimately acquainted with it; and as I am able to refer the reader to the very comprehensive and thorough writings of those authors for the morbid anatomy and pathology of the disease, I shall limit myself here to an epitome and survey of certain aspects of the pathology of beriberi which have a special bearing on the etiological questions that will arise in the course of this chapter.

Clinical history.—The disease,² as we find it, is a complex of a number of symptoms pointing to affections of the peripheral nerves and vessels, which present themselves in the form of disorders of movement and sensibility, particularly in the extremities, of dyspnœa, of scanty secretion of urine, and finally of dropsical effusions. These effusions, however, are not constant; in many cases there are only faint indications of them; sometimes they appear at the very outset of the illness, at other times they are a late addition to the group of symptoms; and thus it is possible to distinguish a paralytic form, a dropsical form, and a mixed form, according as dropsy is wanting altogether or as it comes on late or early. The progress of beriberi is usually chronic; it is rare for it to run an acute course, and when it does so, it is mostly of a pernicious type.

The onset of the first characteristic symptoms is usually preceded for a considerable period (weeks or months) by a well-marked stage of persistent weakness, low spirits, listlessness and unfitness for work. When the outbreak is to take the paralytic form, the onset is shown by a difficulty in moving the lower limbs, which gradually increases to paresis and paralysis; in many cases the upper extremities are attacked with the same weakness, becoming paralysed in the end, and the patient is then placed in a very melancholy situation, being unable to make the slightest movement and requiring even to be helped to his food and drink. At the same time there will have been disorders of sensibility, in many cases commencing as perverted sensations (tickling or creeping feeling, and the like), or as hyperæsthesias (burning, particularly in the feet and legs, the "burning of the feet" of English writers), or as muscular tenderness on pressure (especially in the muscles of the calf).

¹ The authorities quoted throughout the chapter are given in alphabetical order at the end.

² On comparing the accounts given of its symptoms and course by observers in India, the East Indies, Japan, Brazil, and other countries, we find so perfect an agreement in the morbid phenomena that we may regard the process as a whole to have had everywhere the same character; at all events there are no fundamental differences in the type of the disease at the various points of its distribution-area.

Later on the sensory disorder takes the form of anæsthesia, and is specially distinguished by loss of the sense of pressure, and of heat and cold; these symptoms also are noted earliest and most uniformly in the lower limbs, extending afterwards in a smaller proportion of cases to the upper extremities.

Besides these nervous phenomena, there are observed disorders in the blood-making and in the circulation. The patients have the look of anæmia, complaining of palpitation which is not unfrequently very troublesome, and of dyspnœa. Auscultation of the heart discovers blowing murmurs at the apex and over the semilunar valves; percussion shows the area of dulness increased (either from dropsy of the pericardium or dilatation of the heart); the pulse is small, very compressible, slow, or sometimes quickened. The urine is always diminished (in consequence of lowered pressure in the aortic system), but it never contains albumen.

To the phenomena above mentioned, which are characteristic of the so-called paralytic or dry form of beriberi, dropsy is often added in the further course of the malady. In another class of cases dropsy is among the earlier symptoms, and the disease then assumes the so-called dropsical type. Usually the œdema shows itself first in the ankles and legs, extending gradually until it at length becomes general anasarca; then follow dropsical effusions into the serous cavities—always into the pericardium, more occasionally into the pleura or peritoneum, and rarely, as would seem, into the cerebral and spinal arachnoid.

The acute form of beriberi develops either out of the above-described chronic illness; or the disease is acute from the first, with a violent onset of the symptoms, such as has been already mentioned, and with vomiting often added, which, according to some, is specially characteristic and of evil omen. In the worst cases of this variety, the dyspnœa reaches an extreme degree, the patient becomes cyanotic, and dies a few days from the first appearance of the symptoms in a state of asphyxia and with paroxysms of choking. In the chronic form, the issue is not unfrequently towards recovery, the malady often dragging on for months and, with remissions and exacerbations, even for years. Even when the characteristic symptoms are all gone, there are not rarely emaciation and feebleness of the lower limbs remaining over for a long time or even for good. The fatal issue of the chronic form is either by gradual aggravation of the symptoms, particularly the dropsical effusions, accompanied by wasting and general marasmus; or suddenly from asphyxia and paroxysms of choking, as in the distinctive manner of the acute form.

Morbid anatomy.—The anatomical examinations of persons who have died of beriberi have been few hitherto, and they have afforded no certain clue to the nature of disease; or, in other words, they have disclosed nothing uniform in the morbid anatomy.

Among the conditions most commonly found are the following: Remarkably dark colour and extreme fluidity of the blood; serous infiltration of the subcutaneous connective tissue; more or less con-

siderable dropsical effusion into the pleura and peritoneum, but most of all into the pericardium, and now and then into the membranes of the brain and the cord; congestion and often œdema of the lungs; the heart often in a state of "excentric hypertrophy" or simple dilatation, and in many cases soft, pale and fattily degenerated; the inner coat of the aorta and coronary arteries thickened at certain points from fatty degeneration (Lodewijks and Weiss); the brain and spinal cord mostly normal (the softened spots sometimes found in the central organs of the nervous system should perhaps be regarded as *post-mortem* phenomena). In four bodies which they examined, Baelz and Scheube found inflammatory changes in the peripheral nerves, going on to induration and cirrhosis, and similar changes in the muscles—a sub-acute neuritis and myositis reminding us of the cases of multiple neuritis with inflammatory atrophy of muscles, lately described by Eisenlohr, Geoffroy and Leyden, and in which, according to the observers above named, the disease really consists and has its origin.

§ 201. HISTORICAL REFERENCES.

The *history of beriberi* may be followed, so far as the present state of research enables us to decide, as far back as the second century of the pre-Christian era. Inquiries made at the instigation of Dr. Scheube by a number of scholars conversant with the ancient medical writings of China and Japan, with a view to discover whether the malady had existed in those countries, have succeeded in proving that the word "kak-ke" occurs in a Chinese work dating from about 200 B.C., and that there is an unambiguous description of the disease in another work of some one hundred and thirty years later date. Other references to beriberi in China occur in writings of the third, seventh and eighth centuries of the present era; and in a medicine book belonging to the end of the tenth century, there is already a distinction drawn between a "dry" or paralytic kak-ke and a "wet" or dropsical. Of the disease in Japan the earliest record is in a medical treatise of the ninth century of our era; but in that, as well as in subsequent Japanese medicine books, the malady is often confounded with other morbid conditions (heart disease, rheumatism, dropsy¹).

¹ Even in recent times there has been a good deal of confusion between beriberi and the malarial cachexia on the one hand, and the cachexie aqueuse (the anchylostoma-disease) on the other.

For the East Indies the first notice of it occurs in the medico-topographical treatise of Bontius, who was acquainted with the disease there in the seventeenth century under its colloquial name "beriberi." Belonging to the same period is a note upon it by the Dutch physician Tulpius, who had an opportunity of observing the malady in a person returned from India (Coromandel coast). Then follow, in order of time, the accounts by Paxmann, Lind and Fontana, for the Malabar coast, and for the East Coast of the Indian peninsula the important and in some respects fundamental work on beriberi by Malcolmson. Of much more recent date is the appearance of beriberi at various parts of the New World. At what time it first showed itself in the West Indies I have not been able to make out from the accounts before me. In Brazil it was not observed, at least in its more general or epidemic diffusion, until after the year 1860 ; and its first appearance in Guiana would appear to fall about the same time.

§ 202. PRESENT AREA OF DISTRIBUTION.

The *geographical distribution* of beriberi extends over a large part of the tropical and subtropical zones of the Eastern and Western Hemispheres ; but the more important foci of the disease are within comparatively narrow limits. One of its chief seats is the *Empire of Japan*,¹ in which the malady is prevalent from Nagasaki (in the Kiushiu island) to Hakodade (in the Yezo island). Confined until some forty or fifty years ago to the trading ports, it has appeared more recently in the interior as well—in the central provinces of Kodzuke and Oshiu, and even in the mountainous province of Shinano ; and it not only affects the larger towns as formerly but the smaller villages also.² The want of statistical returns of disease in Japan prevents us from forming an opinion as to the amount of beriberi in that country ; but we have an approximate estimate in the number of cases of it returned by physicians in Kioto, which were 2273 from 1875 to 1879 in a population

¹ Friedel, Pompe van Meedervort, Ref. (V), Maget, Warnich, Anderson, Godet, Simmons, Sollaud, Scheube, Bætz.

² Bætz, l. c., 7.

of some 229,000, and of which 1093 occurred in the single year 1878. In the army the number of cases in 1877 was 2687 among a total strength of 19,600 men, or 14 per cent. ; the year after it was 13,629 in a force of 36,100, or 38 per cent.¹ In *China*, where the disease would appear, from the historical data above mentioned, to have been formerly one of the prevalent diseases of the people, it now occurs very seldom.² In addition to the account³ of an epidemic of beriberi in an English regiment in 1852, there is a notice of an epidemic outbreak of it in 1870 in the Cocos Islands (in 12° S. in the Indian Ocean), which came to an end when the patients were sent to the adjoining Keeling Island.⁴ For *Lower India* we have information of its being endemic in Burmah, where it does not appear to have been domesticated until after the outbreak among the British troops in 1824;⁵ also at Singapore,⁶ and in the islands of Pulo-Condo (Calabash Islands) lying off the coast of Cambodia.⁷ On the mainland of Saigon it is very rare, to say the least.⁸ On many islands of the *Malay Archipelago*⁹ it is as widely spread, in endemic or epidemic form, as in the Japanese Empire; thus, in *Sumatra*,¹⁰ the Dutch troops suffered from it severely during the war in Acheen,¹¹ and there are more particular accounts of its endemic prevalence among the natives of the Lampong States.¹² It is found also in *Banka*,¹³ especially in the mining districts; and in *Borneo*, not only in the maritime regions¹⁴ and in the English island of Labuan¹⁵ off the north-western coast, but also in the interior.¹⁶ Also in *Celebes*,¹⁷ particularly in the Government of Macassar,¹⁸ and in several islands of the *Molucca group*, such as Saparua;¹⁹ but in

¹ Scheube (II), 8.² Wernich (I), 293.³ Ref. (II).⁴ Leudesdorf.⁵ Mouat, Kearney, Arokeum.⁶ Ref. I; Ward and Grant. According to Russell, 1174 cases of beriberi occurred in the Singapore gaol from May, 1875, to May, 1880.⁷ Beaufls.⁸ Richard, 'Introd.,' vii. In the medico-topographical writings of French practitioners in Cochin China nothing is said of the disease excepting in the note by Beaufls already quoted.⁹ Schneider, Oudenhoven, Overbeck de Meijer, Swaving, van Leent (I).¹⁰ van Leent (VII).¹¹ Gelpke.¹² Eisinger.¹³ Lindman, van Kappen, van Leent (V).¹⁴ Heymann, Schneider, Rupert.¹⁵ Roe, Barry.¹⁶ van Leent (III, IV).¹⁷ Schmidt Müller, de Meijer.¹⁸ Bauer.¹⁹ Heymann, Robinow.

Ambonia, which used to be very much affected by beriberi,¹ it is now rarely seen.² It exists also on the west coast of *New Guinea*. In *Java* it would appear to be rare on the whole;³ but we have accounts of its endemic prevalence in the Residency of Banjuwangi,⁴ in the extreme east of the island, and in the prisons of Batavia;⁵ also, notices of a severe epidemic in the Residency of Passuruan in 1841, the number of the sick being returned at 8000,⁶ and of epidemics in 1864 and 1865 at the Protestant Orphanage of Samarang.⁷

In *India* the principal seat of the malady is the strip of coast, belonging to the *Madras Presidency*, from Gandjam to Masulipatam, known as the Circars;⁸ from the coast this disease-focus extends with diminishing intensity for about a hundred miles inland.⁹ It is met with very much less frequently on the Coromandel coast,¹⁰ in the plain of the Carnatic,¹¹ and on the Malabar coast.¹² In *Lower Bengal* beriberi became epidemic for the first time in 1877-80 at Calcutta and at various places in the provinces of Dacca and Assam.¹³ There is not a single reference to its endemic or epidemic occurrence on the western littoral within the *Bombay Presidency*, or in the greater part of the *Deccan*, or in the *North-West Provinces*, or in any part of the *Plain of the Ganges*, excepting the above-mentioned epidemic at Calcutta. In *Ceylon* there is a good deal of it, more at some points (Trincomalee, Kandy) than at others (Colombo)¹⁴ and of a more malignant type; but the most recent information that I have of it in that colony dates from 1849, and I am unable

¹ Lesson. ² van Hattem, van Leent (II). ³ Heymann. ⁴ Clapham.

⁵ Ref. (VI); Swaving. From January, 1857, to July, 1870, there were 2069 patients with beriberi in Batavia and on Onrust, who were provided for at the cost of the Government, most of them being in the prisons.

⁶ Brockmeijer.

⁷ van Dissel.

⁸ Marshall (II), Hamilton, Malcolmson, Thomson, Hutchinson, Balfour, Waring, Evezard.

⁹ Kearney informs us that the disease exists in Sumbulpore, Kampti, &c.

¹⁰ For the city of Madras and its vicinity there is not a single reference to beriberi. Huillet says that in Pondicherry he had seen only a few imported cases.

¹¹ Dick.

¹² Wright. According to Day, beriberi is very rare in the Cochin district.

¹³ Fayer, from information supplied by MacLeod.

¹⁴ Hunter, Rogers, Davy, Marshall (I, II), Ridley, Pridham.

to say what may be the state of matters at present. It is altogether doubtful, owing to the absolute silence of the authorities on the point, whether the malady occurs in other parts of the continent of Asia or in the adjacent islands. There is, indeed, a statement by Moore, that beriberi has been seen in sailors at Bassadur (on the island of Kishin, Persian Gulf), but it is most probable that these cases do not imply the prevalence of the sickness on shore but on board vessels frequenting the Gulf. Of which more anon.

In *African territory*, so far as I can learn, the disease has been found hitherto at three points—*Mauritius*, where it was epidemic in 1813 among the British troops;¹ in *Réunion*, where it has broken out epidemically in 1805, 1821, 1838, and 1847;² and on the small island of *Nossi-Bé*, situated north-west of Madagascar, where it occurs from time to time.³

In the Western Hemisphere, as we have seen, beriberi has not been observed until recent times. In the French Antilles, according to a report of Dumont published by Larrey, there are occasional cases of it among negroes and Chinese, which are known as “*maladie des sucreries*,” and there are epidemics of it now and then. It was prevalent on Guadeloupe in 1859 among negroes imported from the Congo Coast. It has been observed also in Cuba,⁴ particularly in 1873, when it raged with great virulence among the negroes on two plantations near Palmira, causing a mortality of from 60 to 75 per cent. of those attacked.⁵ In *Cayenne* it showed it showed itself for the first time in 1865 among imported coolies;⁶ but it does not appear to have recurred⁷ there until

¹ Ref. (III). The epidemic of “acute anæmic dropsy,” which occurred in Mauritius in 1878-79, according to Davidson (‘*Edin. Med. Journ.*,’ 1881, Aug., p. 118), and Pelleran (‘*Arch. de med. nav.*,’ 1881, Avril, p. 298), and which was probably imported from India, is not one that I can regard as beriberi, judging by the symptoms.

² Vinson (I).

³ Guiol. Fayrer’s opinion, that the disease on the West Coast of Africa known as “*negro lethargy*” corresponds to beriberi, rests upon some misconception which I cannot explain.

⁴ Hava.

⁵ Minteguiga.

⁶ Hemeury, Durand.

⁷ The epidemic among the workmen in the gold mines of Sinnarnary, described by Dorvan (Thèse, Montp., 1876), was probably an affair of anchylostoma disease (cachexie aqueuse). At all events it was not beriberi.

October, 1877, when several coolies suffering from beriberi were admitted into the hospital of Cayenne from neighbouring settlements.¹ One of the most considerable centres of the disease has been established of recent years in *Brazil*,² where its general outbreak belongs to the same period as the first cases of it seen in Guiana. Some Brazilian practitioners are of opinion that there had been occasional epidemics before; as at Ceará in 1825 and at Marianna (province of Minas Geraes) in 1858 and 1861. But it was not until 1866 that the general attention of the profession was drawn to it, particularly at Bahia, where there had been sporadic cases a few years before, about the diagnosis of which opinions differed. The point from which beriberi started to overrun the greater part of Brazil was the Bahia province; shortly after, it broke out on the banks of the Rio Anajas in Pará; in 1869 it was in Santa Catharina (according to Rey); at Pernambuco in 1871 (according to Béringer); in the Maranhão province in 1872, Ceará in 1873, San Paolo and Rio Grande do Sul (according to Betoldi) in 1874; as well as in the provinces of Alagoes, Sergipe and Espirito Santo,—thus extending over the whole Brazilian seaboard. It has shown itself also in the interior in other provinces besides Pará, such as Matto Grosso and Minas Geraes; and at many places it has assumed the character of an endemic disease. From the statements of Brazilian army surgeons who had made the campaign against *Paraguay*, it appears that beriberi had been epidemic in that country at the time of the war, at Humaita, Passo da Patria and other places. Lastly, it has to be mentioned that a few cases have lately occurred at San Francisco, but under what circumstances, or among what nationality, does not appear from the very brief notice³ of the fact.

The *epidemic outbreaks on board ship* form episodes in the history of beriberi that are full of interest in several respects. It has been observed oftenest under those circumstances in Dutch men-of-war, transports, and coasting vessels frequenting those parts of the Indian Ocean which encircle the

¹ Hemeury, François.

² Compare the extremely exhaustive work of Férís (I).

³ Ref. (V).

Malay Archipelago,¹ and next to these in British men-of-war and transports in the Bay of Bengal and elsewhere in the Indian Ocean.² There are also a few notices of its occurrence among the crews of ships in the Sea of Japan,³ the Persian Gulf,⁴ and the Red Sea.⁵ In the Atlantic, naval surgeons⁶ have seen cases of it among the crews of Brazilian men-of-war during the war with Paraguay. Guy witnessed an epidemic of it on board a French vessel carrying coolies from Madras to Guadeloupe, the disease having appeared when they were between the Cape and the West Indies and some four weeks previous to their arrival at Guadeloupe. On board another French ship carrying time-expired coolies back from the West Indies to India, beriberi broke out among the coolies when they had got as far through the Indian Ocean as 91° E. and 14° N.⁷ Among other instances of epidemics on board coolie ships from India to the French Antilles, or returning, are those of the *Nicolas Poussin* in 1864 and the *Marie Laure* in 1878. One of the worst cases is that of the *Parmentier* in 1861-62; she was carrying 401 coolies back from Martinique to Pondicherry, and after a voyage of five months she landed only 281 coolies alive. In ships navigating the Southern Ocean or Australian waters,⁸ beriberi has never occurred, so far as I know.

§ 203. INFLUENCE OF CLIMATE AND SEASON.

As regards the *manner of its occurrence*, beriberi has at some places within its distribution area the character of an

¹ See the accounts of Schneider (l. c., 14), Heymann (II), Pop, van Leent (I, III, IV), Steendyk, Zuur, Schutte, Westhoff, Rupert.

² Hunter, Carter, Morehead (I).

³ Anderson.

⁴ Moore.

⁵ Wellstead, Pruner, Carter.

⁶ Ribeiro de Almeida, 'Estudo sobre as condições hygienicas das navios encouraçados.' Rio de Janeiro, 1871; Saraiva, 'Quaes os melhores meios de combater o beriberi?' Bahia, 1871. (Quoted by Férís.)

⁷ Richaud.

⁸ The outbreak of an "epidemic dropsy," described as having occurred in 1856 on board H.M.S. *Juno*, while cruising off the Australian coast ('Statist. Rep. of the Health of the Navy for the year 1856,' p. 161), is, in my opinion, referred to beriberi on insufficient grounds.

endemic malady, as in many islands of the Malay Archipelago in the northern division of the Madras Presidency (Circars), in Ceylon, at Singapore, in the Japanese Empire, and in many parts of Brazil ; whereas at other places it shows itself only in *epidemics* separated from one another by more or less considerable intervals of time. To the latter class its outbreaks on board ship belong very characteristically.

The *influence of climate* does not account for these differences, any more than it accounts for the existence of the disease at all. No doubt the area of beriberi is almost exclusively within tropical and subtropical regions ; but the endemicity of the disease at Hakodade (Yezo), with a climate nearly corresponding to that of the temperate zone of Europe and America, and the outbreaks on board ships in somewhat high latitudes, are evidence that the malady may develop or persist, even as an endemic, beyond those regions. That the tropical or subtropical climate does not of itself determine the endemic or epidemic existence of the disease, follows from the fact that the several centres of beriberi are often narrowly circumscribed within its distribution area, while the regions nearly adjoining them and subject to the same climatic influences escape the disease ; as well as from the fact that it is quite unknown in Arabia, on the East and West Coasts of Africa, and in other regions with a pronounced tropical climate ; and that in Guiana and Brazil, it is only recently that it has, I will not say occurred, but at all events become general, although we cannot account for that by anything in the climatic conditions.

But we must none the less admit that the development of the disease into an epidemic stands in very definite relation to the influence of *season*, or of the corresponding *weather conditions*. Observers in all parts of the world where beriberi has been observed hitherto are unanimous in saying that the largest number of cases, or the epidemic outbreaks, fall in the season which is meteorologically characterised, first of all by the high degree of atmospheric moisture, and next by great vicissitudes of temperature. This is the opinion expressed by Bontius, v. Oudenhoven, Heymann, Schneider, v. Dissel, Rupert and others for the East Indies ; by Hamilton, Malcolmson, Waring, Kearney, and others for

the Circars ; by Dick for the Carnatic ; by Lind, Fontana, Wright and Day for the Malabar coast ; by Marshall for Ceylon ; by the authority for Singapore ; by Wernich, Simmons, Scheube and Baelz for Japan ; and by da Silva Lima, Pacifico Pereira and others for Brazil. Proof of this is afforded by the statistics of sickness given by some of the observers.

The following table shows the distribution according to season of 572 cases of beriberi, admitted into the military hospitals from among the native troops in the northern division of the Madras Presidency during a period of three years.¹

Table of Admissions for Beriberi in the several Seasons.

Hot Season.	Rainy Season.	Cool Season.
March . . 19	July . . 34	December . 54
April . . 27	August . . 68	January . . 25
May . . 13	September . 99	February . 17
June . . 27	October . . 90	—
—	November . 99	96
86	390	
or 15·03 per cent.	or 68·2 per cent.	or 16·7 per cent.

The cases at Tokio from 1879 to 1881, to the number of 2224, were distributed as follows:²

January . . 37	May . . 212	September . . 194
February . . 23	June . . 341	October . . 64
March . . 39	July . . 632	November . . 17
April . . 124	August . . 537	December . . 4

The approach of the cold and dry season has almost always had a good effect on the type of the attack, as well as in bringing an epidemic to an end. Observations as to the weather at the time of an epidemic outbreak, bearing out those above mentioned, have been repeatedly made at sea by Hunter, Lindmann, Richaud, and others.

¹ Waring.

² Baelz.

§ 204. INTIMATE ASSOCIATION WITH THE SEA COAST.

Whether the occurrence of beriberi, and its diffusion, are independent of particular *kinds of soil* cannot be decided with any approach to certainty. One noteworthy fact is, that the malady has been mostly prevalent on the sea-coast, the shores of great rivers, and the plains adjoining; while it has shown itself seldomer in the interior, and has for the most part avoided high tablelands and mountainous country. Hamilton, Marshall, and others among the earlier observers, were of opinion that a distance of forty to sixty miles from the coast was sufficient to give immunity from beriberi; and several of the more recent authorities have expressed the same opinion. Malcolmson found this idea confirmed by his experience in the Circars; but he thought it advisable to add that “this law will, I have no doubt, require to be greatly modified as our information is extended”—a wise reserve which has been justified by subsequent experience. As early as Balfour’s report on the cases of beriberi among the troops in the Madras Presidency from 1829 to 1838, we find considerable exceptions to the rule in question. The cases to the number of 1116 were distributed as follows:

Stations on the coast	394
Stations on the plain, between eight and forty miles from the coast	537
Stations on the plain, between forty and one hundred miles from the coast	132
Stations on the high ground, more than 1300 feet above the sea	53

It should be kept in mind, also, that there were only two stations of the last kind, viz. Secunderabad and Kampti; and that the troops quartered in them were a comparatively small force.

But we find still better evidence that the area of the disease is by no means absolutely associated with the conditions obtaining on coast-lands, in the latest experiences from those same districts of India (Kearney), from Assam (Fayrer), and from Burmah (Kearney), where beriberi has occurred hundreds of miles up country; also in the observations on

its extension in Japan, from the coast into the interior; and, most of all, in the fact that it has been found to be epidemically prevalent in one of the western provinces of Brazil. Therefore, although the preponderance of beriberi on the coast is still unmistakable, the explanation of the fact lies perhaps more in the meteorological conditions than in any peculiarities of soil.

With reference partly to the preponderance of beriberi on sea-coasts and river banks, some¹ have laid special emphasis in the etiology upon the *wetness or swampiness of the ground*, and have deduced therefrom the conclusion that the disease partakes of a malarial character. I shall afterwards show that beriberi has not the very smallest point in common with malarial diseases. For the present, I shall merely meet the theory of an influence derived from wet or swampy soil by directing attention to the fact, that the principal seat of the malady is that very region of India which is comparatively well off as regards the moderate saturation of the soil; whereas, the regions that are most damp and most abounding in swamps, such as Orissa, the plain of Lower Bengal, and the plain of Cambodia are almost exempt from beriberi. Further, that the disease has risen to a more or less important place within the last thirty or forty years at various points, both in the Eastern and Western Hemispheres, where it used to be either rare or altogether unknown, while there have been no concomitant changes in the soil; that beriberi is met with much more commonly in towns than in country districts; and last, but not least, that it can spring up and become epidemic on board ship, where there can be no question of an influence of soil in the ordinary meaning of the term.

§ 205. OCCURS MOSTLY IN ADULT MALES, NATIVE AND
ACCLIMATISED, AND IN THOSE OF GOOD PHYSIQUE.

Inquiring into the influence upon the production of beriberi exerted by things prejudicial in the mode of living, we

¹ For example, Swaving, Bary, and Rupert for the East Indies; Anderson and Simmons for Japan; Betoldi, Rey, and Pereira for Brazil.

have in the first instance to consider as factors in the etiology a number of personal things, such as age, sex, nationality and constitution.

As regards the *amount of the sickness at various ages*, there is absolute unanimity among the authorities that cases of beriberi are the exception in children or young persons under the age of fifteen, no such cases being met with in many of the epidemics; that old age is also exempt for the most part; and that most of the cases are in people in the prime of life. On board the ship *l'Indien* the emigrants numbered 575, among them 54 children up to fifteen years of age; there were 118 cases of beriberi, but only one of these was in a child (Guy). On board the *Jacques Cœur*, the whole of the 55 children of the coolies escaped the disease, while of the 332 adults, 44 took it (Richaud). In the epidemic of 1869-70 in Labuan, the age of childhood was found to be quite protected (Barry). Of 35 fatal cases in the hospital of Tokio, only one was in a person under fifteen; of 581 beriberi patients treated by Scheube at Kioto, only 35 were children; of 933 patients at Tokio, 15 were between ten and fifteen years of age, 753, or 80 per cent., were between sixteen and thirty years, and the remaining 165 cases were over thirty years, 89 of them being between thirty and forty. That the rule is not without exceptions, is proved by the epidemics occasionally observed in orphanages and training institutions. Thus in the Protestant Orphanage at Samarang in 1864 and 1865, there were 98 cases among 235 children, and 9 of these fatal; and in the Missionary Schools at Toukiji in Japan, a large number of girls between the ages of ten and sixteen were attacked with beriberi, of whom one died.

A difference in the liability to beriberi, not so pronounced perhaps as that which distinguishes childhood from the prime of life, but still a considerable difference, exists *between the female sex and the male*. In Ceylon, Christie did not see a single case in a woman, and Rupert says the same for Borneo. According to Heymann and others for the East Indies, Wernich and Simmons for Japan, Pereira, Betoldi, Rey and others for Brazil, and Larrey for the West Indies, the cases of beriberi in the female sex are rare. On board the

emigrant ship *l'Indien*, there were 111 cases among 385 men, or 29 per cent., (of whom 40, or 10·4 per cent. died), while there were only 6 cases among 118 women, or 5·5 per cent., of whom 2 died (Guy). But on board the coolie ship *Jacques Cœur*, the proportion of cases among men and women was as 12 to 7. Among 584 patients with beriberi, Scheube gives only 50 females; and among 2224 cases treated in the hospital of Tokio from 1879 to 1881, there were, according to Baelz, only 68 women, or 1 to 31·7 men. Agreeing with Simmons, Baelz is of opinion that pregnancy and childbed make the liability considerably greater.

A third personal factor in the pathogenesis consists in the *nationality* of the individual. At every endemic or epidemic seat of beriberi where there is a mixed population, it is mostly the natives or immigrants belonging to the dark races who are attacked, the Europeans and Americans enjoying, particularly in the first period of their residence, an immunity which, if not absolute, is still a very pronounced one. In the East Indies and on board the ships of war on the Dutch East Indian Station, according to the unanimous opinion of v. Oudenhoven, van Leent, Praeger, Rupert and others, the malady occurs principally among natives. Only in exceptional instances has it been seen to any considerable extent among Europeans, as in Amboina among very poor people, according to van Hattem, and on board a Dutch man-of-war, where the epidemic, according to Heymann, was strictly limited to the Europeans of the ship's company. The extent to which the malady is prevalent among the Malay crews of the Dutch East Indian squadron, is shown in van Leent's statistics¹ for the years 1870-77, according to which the proportion of cases in Malays and Europeans was as 60 to 1. In the earliest accounts of beriberi in India, those by Fontana and Lind, mention is no doubt made of somewhat frequent cases in Europeans; but all the more recent authorities are agreed that the latter suffer very much less than the natives (and than Mohammedans, in particular, according to Malcolmson). From 1829 to 1838 there were only two cases of beriberi among the European troops in the Madras Presidency (Balfour); Waring estimates the proportion of native cases

¹ 'Geneesk. Tijdschr. voor Nederl. Indie,' 1880, ix, 297.

and European as 6 to 1; during the epidemic of 1878-80, in Calcutta and its suburbs, there was not a single case in a European (Fayrer). In Ceylon, Marshall has seen beriberi only in the blacks; and Ridley, for the same colony, says that Europeans are certainly subject to the disease, but much less commonly than the natives. In Japan, the Europeans and Americans enjoy an almost absolute immunity; among two thousand foreigners in Yokohama, Simmons could learn of only one well-authenticated case in a European; in the foreign colony at Tokio, Wernich saw only two cases, one in an Italian and the other in an American woman; according to Anderson, not a single case of beriberi had occurred among the British and French troops formerly stationed at Yokohama; in his own experience Scheube knows of only two authentic cases in Europeans; and Baelz says that Europeans in Japan hardly ever take beriberi. In the West Indies, according to Larrey and Minteguiaga, the malady has been seen hitherto only in negroes and Chinese; in Guiana, according to François and Hemeury, only among imported coolies, with the exception of a case which the latter saw in a European sailor. In Brazil, also, the accounts given by practitioners show that the natives suffer much more than the foreign immigrants.

It is a noteworthy fact, already adverted to, that the predisposition to an attack of beriberi *increases* with the length of time spent at the focus of disease, or with the *degree of acclimatisation*. Calhoun¹ was the first to point this out when he wrote as regards Ceylon: "It would appear that a stay for some months on the station is almost essential for the production of the disease, and that the greatest predisposition to it exists when troops have been about eight or twelve months in the settlement.

Hamilton and Malcolmson afterwards took the same view, from their experience in the Circars (Madras). Subsequently the fact was confirmed by observations made in Japan. Simmons' statement is that among the Japanese who have come from the interior to the coast (the proper seat of the malady), it never appeared until after they had resided some time; but that then the cases became more frequent among

¹ Quoted by Hunter, l. c., p. 96.

them than among natives of the coast (in which opinion Baelz agrees) ; and that therein lies the explanation of the large number of cases among sailors, soldiers, policemen, students and others, who had come to the seaports from the interior. The period required for acclimatisation, or, in other words, for becoming predisposed to the sickness, is estimated by Baelz at from a few months to a year ; and the same period is fixed by practitioners in Brazil, where strangers are exempt from attacks of beriberi for six or twelve months after they have come to reside at one of the indigenous centres of the disease. One attack makes a greater liability to others ; so that those who have suffered from beriberi once may have attacks recurring at regular intervals for years after, as Hamilton had shown to be the case in India, and Wernich, Scheube and Baelz in later times have proved for Japan.

The last consideration in the question of individual predisposition, is that in the experience of the East Indies, Japan and Brazil, people of *strong physique* take beriberi much oftener than the weakly. "Evidence that it is not the weakest who take the disease soonest and most severely," says van Overbeck de Meijer, "is furnished by the experience of the new State Prison of Batavia, during the epidemic of beriberi ; for it was just the strong people who took the disease and died." The same fact has been established by Lindmann for Banka, by Mohnicke for Amboina, and by Rupert for Sumatra. "So far as relates to constitution," says Rupert, "the fact was brought out that there were no doubt a few weakly individuals among the patients, but that the great majority were strong persons between the ages of twenty and thirty, and that it was often actually the strongest and best nourished who were attacked." To the same effect are the accounts from Japan by Simmons and Baelz : among 626 out-patients treated by the latter for beriberi at the hospital of Tokio in 1881, there were 593 of robust constitution, 27 of moderate strength and 6 weakly persons. Such is the view, also, of many of the authorities in Brazil ;¹ among others of Caire, who says that all the cases of beriberi which he had to treat at the Sailor's Hospital of Rio, were in strong, athletic individuals. It is only the

¹ Férís, 'Arch. de méd. nav.,' 1882, Juin, p. 476.

previous attacks of exhausting diseases, such as dysentery and protracted agues, that appear to increase the predisposition to beriberi; and that is perhaps what van Leent (I) refers to when he says that invalids are particularly liable to it.

§ 206. SEDENTARY OCCUPATIONS PREDISPOSE TO IT.

With a special exception in favour of the very numerous outbreaks of beriberi among bodies of troops and on ship-board, particularly in men-of-war and transports, the various classes and callings of the population would appear to be subject to it somewhat uniformly; at all events no class enjoys immunity by virtue of its wealth or its social position.

Féris in his summary of the Brazilian writings on the subject says: "On voit quelquefois l'affection attaquer les personnes qui sont dans une position élevée; on peut dire que, jusqu'à présent, aucune position sociale n'a été respectée." The accounts are the same from Japan, where, as Baelz says, people in comfortable circumstances are attacked actually oftener than the working classes and the proletariat. Scheube is of the same opinion, and he adds that even the upper and very highest ranks of Japanese society are not exempt from beriberi; according to his observations, the great bulk of the patients is made up of scholars, priests, teachers, pupils, merchants, artists and handicraftsmen. Of 333 male patients treated by him in 1877 and 1878, there were 261, or 78 per cent., belonging to those callings; and of these again, 168, or 46 per cent., were scholars, priests, teachers, pupils or writers, while 106, or 41 per cent., were in business, and 37, or 13 per cent., were artists or artificers. The conclusion which Scheube draws is, that those occupations predispose specially to the disease which involve a sedentary life;¹ and that is also the conclusion which the Brazilian practitioners have come to, according to Féris, when he says: "Signalons

¹ This fact disposes of the suggestion thrown out by v. Leent ('Geneesk. Tijdschr. voor Nederl. Indie,' 1880, ix, 306), that the comparative freedom of the female sex from beriberi may be due to their having less physical exertion to undergo than men.

la vie sédentaire comme une cause secondaire de la maladie ; c'est pour cela, sans doute, qu'elle se développe si facilement chez les individus qui appartiennent à la classe lettrée."

§ 207. INFLUENCE OF OVERCROWDING AND WANT OF VENTILATION.

It seems to me to be extremely doubtful whether the want of physical exercise is in itself a factor in the etiology, inasmuch as soldiers and sailors, who are particularly often attacked by beriberi, have certainly no lack of exercise. A much more likely suggestion is that *long confinement in more or less crowded and badly ventilated places*, so often associated with the mode of life of those classes, plays a leading part in the production of the disease ; and that is the opinion held by many of the authorities. Noteworthy in this connexion is the exceedingly frequent occurrence of beriberi in the gaols of British India and the Dutch East Indies, as well as in schools and boarding institutions, the defective ventilation of which has been specially dwelt upon by van Dissel in treating of the epidemics of Samarang ; and his observations are all the more deserving of attention for the reason that they relate to young girls who are not often subject to beriberi under other circumstances. The same is true of the outbreaks of the disease on board transports and coolie-ships ; for Hunter, Guy, Richaud and others are agreed in saying that the one obvious cause of the outbreaks has been the insufficient ventilation of the 'tween decks occupied by the troops or emigrants, the want of fresh air becoming all the more pressing when the hatches had to be kept closed for a length of time in consequence of bad weather. Swaving, also, with whom many observers in India agree, lays special stress in the etiology of beriberi, upon prolonged and frequently recurring confinement in small, overcrowded and ill-ventilated places, more particularly if they be used also for sleeping in ; and he shows, from facts observed, that the degree to which the disease develops is in proportion to the seriousness of the nuisance in question and to the duration of it. In like manner Rupert is of opinion that "fouling of the air by

noxious matters, such as investigation cannot discover with precision," is one of the more intimate causal factors; and Scheube concludes from the facts before him that "persons who live together in large numbers in small rooms are quite peculiarly liable." In this we may find a partial explanation of the frequent occurrence of beriberi among the crews of men-of-war, and among troops in garrison.

§ 208. EVIDENCE OF A DIETETIC CAUSATION.

The point most keenly debated in the etiology of beriberi is the question of the influence of *deficient or improper food and drink* on the pathogenesis. As in all such cases of obscure causation, there has been no lack of assertion that the true and proper cause of the malady is to be looked for in the use of *tainted or brackish drinking water*. This doctrine, which was put forward first by Wright and afterwards brought into currency by Evezard, has been contradicted by the experience of other observers (Malcolmson, Richaud and others); and, according to the most recent facts from Japan and Brazil, it has now lost all importance.

It is otherwise with the question of an influence exerted by an *insufficient diet or a diet not corresponding to the metabolisms and bloodmaking, or to the needs of the body*; such would be the exclusive or preponderant use of rice, which is especially poor in nutritive matters, and of dried fish (deficient in albuminous substances and fat); and these are in many parts of Eastern Asia the principal articles of food not only among the lowest of the population but also in the middle classes. The Anglo-Indian physicians had at an early period pointed to this harmful influence as a cause of the malady; and by many of the recent observers in the Dutch East Indies and in Japan it has been assigned the first place in the etiology. "Whenever beriberi appears among a ship's company," says Overbeck de Meijer, "it is always in consequence of their having to live exclusively on salt meat owing to circumstances beyond our control; and whenever the troops employed in military expeditions in certain parts of the Dutch East Indies are obliged to live

exclusively upon the ordinary food of the country, they are almost always attacked by beriberi;" and Pop had previously spoken in the same way of the disease as it occurs on board Dutch ships-of-war on the East Indian station. Van Kappen draws attention to the fact that those of the Chinese employed in the mines of Banka who live well, are exempt from beriberi, and that the persons attacked by the malady are those who become anæmic in consequence of insufficient food. Stendijk's opinion was that beriberi appeared on board ship whenever the food ran short or became spoiled. A similar view is taken by Westhof, who complains of the bad provisions (rice and dried fish) supplied by the river-passenger companies to their vessels in the Dutch East Indian service; and by Schutte, who says that he had seen the disease in the gaol of Paramaribo under the same circumstances (diet of rice, salt fish, and boiled green bananas). Not less decidedly has van Leent adhered to that opinion on every occasion when he has had to advert to the occurrence of beriberi in the Dutch East Indies, and at greatest length, in his latest article on the subject.¹ "The dietetic error," he says, to translate literally, "which I regard as the one and only cause of the morbid composition of the blood in beriberi, consists in the too small proportion of albuminous substances and fat." In proof of this he gives the experiences of the disease in the Dutch East Indian fleet from 1870 to 1878, particularly during the war in Acheen, both among the native and European members of the crews. Until 1873 there existed, in consequence of the extremely inadequate rations of the natives, that marked difference between the number of cases in them and in Europeans which has been already referred to (p. 584), a difference that was represented in 1873 by the ratio of 60·37 to 0·88. From 1874 onwards the Japanese sailors got the same rations as the Europeans, whereupon the proportion of sickness from beriberi among the two races became as 7·06 to 0·07. "I can say positively," he adds, "that there was not the slightest alteration in any of the circumstances, apart from the radical change in the rations supplied to the native part of the crews." This experience confirms van Leent in the

¹ 'Geneesk. Tijdschr. voor Nederl. Indie,' 1880, ix, 295.

conclusion that he had come to from former observations, a conclusion formulated by him in these words:² “Le bérubéri reconnaît comme cause principale une alimentation trop uniforme, insuffisante et de mauvaise qualité; l'organisation, privée des éléments indispensables à l'entretien de la composition normale du sang et par suite à la nutrition, s'appauvrit peu à peu.”

The same view of the influence of improper diet in producing beriberi has been taken up by Maget and Wernich, two of those who have studied the disease in Japan. “The kak-ké,” says Wernich,² “is a chronic constitutional disorder of blood-making and of the vascular system. Rice as the exclusive food of the people is answerable for it in a quite especial way. Not, however, as some have thought, because it is used in a decomposed state, but because it is used in such quantities that the power of assimilation is gradually lost for other kinds of food; and even the large quantity of rice is unable to render the nutrition and blood-making adequate. Although the Japanese diet contains albuminous elements in the form of fish and bean cheese,³ these are not sufficient. Fat in an easily digestible form, as the Northern Chinese have it in considerable abundance in their fat pork, mutton and duck, and the Southern Chinese and Malays in palm-oil, is almost wanting in the diet of the Japanese.”

To this reading of the importance of certain deficiencies or errors in the diet for the development of the malady, considerable objection has been taken; and, in so far as these objections are directed against the exclusiveness of the doctrine, I think them entirely justified. Malcolmson, criticising the earlier views on the influence of diet held by Anglo-Indian physicians, wrote as follows: “Much has been said of the effects of various kinds of food, and Dr. Herklots enumerates a number of articles, whose use he considers injurious; but when we reflect that these are standard aliments all over India, we cannot carry our deference to his experience so far as to admit that they can produce, in these districts only, so singular a train of symptoms. What effect

¹ ‘Arch. de méd. nav.,’ 1867, l. c.

² ‘Geographisch-medicinische Studien,’ p. 193.

³ The Japanese “tofu,” a kind of porridge made from old and dried beans, and consisting mostly of the legumin. (See Wernich, l. c., p. 85.)

the extensive use of fish may have, in combination with other influences, I am not prepared to say ; but the comparative cheapness of all kinds of grain in the Circars, and the easy circumstances of many of the native soldiers who suffered, are fatal to any supposition of the disease depending on deficient and unhealthy diet."

The slight influence of deficient diet on the production of the malady is further shown in the fact stated by Waring, that during the frightful famine which visited the South of India in 1833 and 1834, not a single case of beriberi was observed in those gaols of the Madras Presidency which had been free from the disease before, although it was epidemic during that period in the gaols of Bellary and Cuddapah. In the account of the epidemic of 1878-80 at Calcutta, drawn up by Fayrer, it is stated that the disease was prevalent mostly among the poorer classes of Hindus and Mohammedans, but that Eurasians and well-to-do natives were by no means exempt from it, and that dearness of provisions or improper diet could by no means be made out to be causes of it, inasmuch as well-fed persons and flesh-eating Mohammedans suffered along with the poor.

The observations of Rupert in Borneo form a specially interesting contribution to the discussion of this matter. When beriberi was epidemic among the troops and the crews of men-of-war, these had twice a week fresh beef, poultry, eggs and coffee in their rations besides fish, salt meat, potatoes and rice ; at the same time the labourers of the country, whose food for long periods was nothing but rice and a piece of dried fish, were entirely free from the disease. Rupert's opinion is very much that of Malcolmson. "If the notion were correct," he says, "that an improper diet, or a preponderance of amylaceous and vegetable food and deficiency of animal, plays the chief part in the production of beriberi, the disease would have to be a very general one, inasmuch as rice is the staple diet of the people of India and the East Indies, remarkably little animal food being used by the natives excepting in the case of a few hunting tribes in the interior of Borneo and Sumatra. But the fact is that it is restricted in the Dutch East Indies to certain regions and spots, mostly on the coast or in immediate proximity to it."

Simmons, who admits that rice is an inadequate kind of food, ill suited to anyone suffering from beriberi, expresses a doubt whether the excessive use of it is to be held as the cause of the malady in Japan. Scheube and Baelz enter the lists very decidedly against that doctrine; the former pointing to the existence of the disease among the hunters and fishermen of the Ainos (in Yezo), whose diet is far superior to that of the Japanese in albumen and fat; and the latter pointing out, as Rupert had done for the East Indies, that in Japan the disease is commonest where there is most animal food in the diet, that is to say, at the sea-side.

Finally, Brazilian authorities are most decided, and almost unanimous, in disputing all connexion in the pathogenesis with deficient or improper diet, for the reason that beriberi is common among the proprietary classes of that country, surrounded by all the comforts of living. "*Comment se fait-il,*" asks Féris, in his summary of the facts about beriberi in Brazil, "*que les individus les plus atteintes soient ceux de la classe élevée plutôt que ceux de la classe inférieure qui, luttant avec la misère, s'alimentent mal ou insuffisamment, et pourtant sont précisément ceux qui payent le moindre tribut à l'épidémie?*"

In the Brazilian fleet it was epidemic on one occasion, when there was actually a superabundance of provisions. Guy and Richaud also state that there could be no question of short rations or bad provisions in those instances of beriberi outbreaks which came under their own notice on board French transports. One more proof that beriberi, in its endemics or epidemics, is altogether independent of this etiological factor, I find in the circumstance that there has been no general diffusion of it in Brazil until recent times, while there has been no such change in the staple food of the people as would render its phenomenal outburst intelligible.

§ 209. VARIOUS THEORIES OF THE NATURE OF BERIBERI.

Impartially summing up the results of the foregoing inquiry into the connexion between beriberi and certain con-

ditions of climate, weather, soil, constitution, mode of living, and of diet, we find nothing which supplies an answer to the question. Where are we to look for *the true and proper cause of the disease*? What we do learn is that it must be something over and beyond those influences. The difficulties which we encounter in trying to solve this question are considerably increased by the obscurity that surrounds the nature of the disease itself, an obscurity which the most recent observations and researches have not dispelled. At the present day, just as in the earlier part of the century, the most various opinions obtain as to the nature of the disease, and as to the disease-producing factor; so that we can very aptly express the present standpoint of our knowledge in the phrase, "*autant d'auteurs, autant d'opinions diverses.*" It would be beyond the limits of my task to go at all deeply into the disputed *nature of beriberi*; I must therefore content myself with touching upon it only in so far as it affects our critical estimate of the opinions held by various authorities in regard to the causation.

One of the oldest theories starts from the point of view that beriberi is a *rheumatic process*; or, to put it more generally, a chill-disease brought about by hot and damp weather with great fluctuations of the temperature. To this doctrine Fériss¹ has lately given his adhesion; he denies, indeed, the rheumatic character of the disease (in the ordinary meaning of the term) as well as its specificity ("*entité morbide*"); but he takes the "*cause déterminante*" to be "*l'influence des phénomènes météorologiques, à savoir: chaleur humide et transitions brusques de température,*" and the "*cause prédisposante*" to be "*affaiblissement des vaso-moteurs et du grand sympathique.*" This theory of the origin of beriberi, like every other theory which would refer the disease to a cause in general operation, without taking into account the fact that the malady is confined within small and definite areas, is, in my opinion, to be set aside as erroneous on the face of it. Even if we admit, in consideration of the facts above given, that states of the weather are not without an influence on the development of the disease, and more particularly that the frequent occurrence of beri-

¹ 'Arch. de méd. nav.,' 1882, Août, l. c.

beri on board ship and among troops is connected therewith ; we should still be unable to understand how it is that a harmful influence of so common a kind as that sort of weather, distinctive as it is of the climate of many tropical and sub-tropical regions, should give rise to outbreaks of disease of a most definite and peculiar type at only a few points on the globe, and these far apart ; why in India, for example, where the meteorological conditions in question make themselves felt with equal force over a great part of the country, the spots of beriberi should be so few, and confined within so short a radius. Furthermore, the existence of " *affaiblissement du grand sympathique* " is an assumption for which there is not the smallest warrant in the previous histories of the patients, the larger number of whom were of strong physique and otherwise in good health.

The view entertained by Christie, Morehead, Carter, van Overbeck de Meijer, Praeger and others, that beriberi is a disease closely allied to scurvy, is based on the fact that the two diseases have sometimes been prevalent together, and on the assumption that the morbid causes of both belong to the same group, namely, defects or errors of diet. Against this doctrine it has to be said that a coincidence in time observed now and then between two diseases, does not warrant a conclusion as to their general identity ; that a comparison of the aggregate symptoms peculiar to each disease does not bring out the smallest resemblance between them ; and that the food-factor in the genesis of beriberi plays, as we have seen, by no means so conspicuous a part as to warrant us in explaining the production of the malady by reference to it alone.

The same objection has to be taken to the theory first put forward by Evezard and Lodewijks, afterwards stated more precisely by Wernich and adopted in its later form by Schutte, the theory, namely, that beriberi is a *kind of pernicious anæmia*, or, as Pacifico Pereira puts it, that it is a " *dystrophie* " depending upon insufficient oxidation of the blood, induced by a variety of debilitating influences, and having its real cause in errors of diet. It seems to me to be beyond question that a state of anæmia brought on by disordered nutrition is an essential part of the morbid

phenomena ; but it is still a doubtful point whether this abnormal nutrition constitutes the primary factor, the starting point of the malady,¹ or whether as Simmons thinks, it is a secondary development in the course of the disease. Certainly beriberi occurs both endemically and epidemically, where no error of diet can be discovered as the morbid cause ; and on the other hand, poor diet in every possible form and with all its consequences has been common in all ages and in all parts of the world without any epidemic or endemic of beriberi developing from it. Those who would cling to the idea that this etiological factor is the true cause of the malady, will have to find evidence that the bad food acts in some such peculiar and specific way as would account for the specific character of the disease ; but this has not been done as yet in a satisfactory manner.

Lastly, beriberi has not escaped the fate of being counted, among the *malarial diseases*, in the opinion of many observers such as Heymann, van Hattem, Swaving, Clapham, Roe, Barry, Russell and others in India, Ceylon and the East Indies, of Simmons in Japan, and of many of the authorities in Brazil.² The evidence for this consists partly in the prevalence of both diseases together in endemics or epidemics, and in the fact that persons who had suffered from malarial fever sometimes take beriberi ; and it is partly derived from the occurrence of beriberi on wet or marshy soil. Apart from the consideration that there are scarcely two other diseases which show so great differences in their type and evolution as beriberi and malarial disease (the enlargement of the spleen, which is characteristic of the latter, being never found in beriberi), and that the coincidence of two diseases in time or place, (far from constant, moreover, in the present instance,) is not of itself enough to show their identity, there is the objection to be taken above all others to this theory that the most intense foci of malaria in India, although they immediately adjoin the beriberi districts—I have specially in mind Orissa and the delta of the Ganges—are

¹ v. Leent, who holds this opinion ('Geneesk. voor Nederl. Indie,' l. c., p. 307), expresses himself very decidedly against identifying the anæmia of beriberi with the so-called "pernicious anæmia."

² See Férís, 'Arch. de méd. nav.,' 1882, Août, p. 83

absolutely free from the latter disease, that the coast of China, which is one of the chief seats of malaria, is but slightly affected with beriberi, that Davy, although he inclines to the view of beriberi being a form of malarial sickness, did not meet with a single case of it in Ceylon during four years medical service there, and that beriberi has never been seen in the great malarious countries of tropical Africa. It is a hardly less telling fact against the malarial theory that beriberi is endemic in many regions where malarial disease is rare, as at Singapore and at a number of mountainous places in the East Indies; and still more decisive facts are that epidemics of beriberi may develop on board ship, that women enjoy an exemption, and that the non-acclimatised are spared, although they are the very subjects to suffer most from the influence of malaria wherever it is endemic.

§ 210. THE CAUSE OF IT A SPECIFIC ONE.

That beriberi is a specific morbid process, a disease *sui generis*, is proved, not only by the *ensemble* of symptoms and the whole type of the malady, which presents no clear analogy to any other disease known to us, but also by its history in place and time, by its epidemic outbreaks, and by its establishment as an endemic. From these facts we must conclude that there is a peculiar and *specific cause*; and inasmuch as the specific cause is not discoverable, according to the present state of our knowledge, either in conditions of climate, weather and soil, or in the general manner of living and dieting among the inhabitants of the affected parts of the globe, it must reside, as we have already indicated, in some pathogenetic influence over and above these latter, which cannot themselves be made out to be of importance for the development of the disease except only in so far as predisposing or affording an opportunity. But although that assumption appears to be justified, no one has succeeded as yet in coming to any definite conclusion on the nature of the "morbid poison."

The conjecture that the morbid poison is some *specifically noxious thing in the food*, brought about by the local condi-

tions, is a probable one, and there has been no lack of hypothesis in that sense. At an early period the question was propounded, whether it might not be some *poisonous property of (decomposed) rice* that represented the cause of the malady ; and that doctrine has recently found a defender in Kearney, who, like Malcolmson at an earlier date, points to the resemblance in particular symptoms between beriberi and ergotism, although he brings forward no other positive evidence to support his opinion.

Other observers think that the disease is of *parasitic* origin. This hypothesis, worked out by Gelpke, makes a very odd impression, and I give it in the author's own words :

"It is only a living poison, such as can lie long latent in the body, and, perhaps, undergo sexual development therein, that can generate beriberi; and in this sense I may compare beriberi with *trichinosis*. Let no one misunderstand me, however. I claim for this beriberi-trichina neither the size nor the sexual natural history of *Trichina spiralis*. But the host of the parasite, at all events, is the dried fish upon which prisoners are fed. These fish do not live in the waters of the Malay Archipelago . . . and I have ascertained as a matter of fact, through my inquiries on the subject, that the fish is imported from China. . . . The diffusion of the disease extends to every place where the fish is caught and eaten; it breaks out in epidemics wherever the fish comes in its migrations . . . or wherever its poisoned flesh is introduced by commerce."

The evidence that Gelpke brings forward to support his theory is little likely to create confidence in it. The same remark applies to the writings of Erni, who believes that he has discovered the cause of the malady in an affection of the intestinal mucous membrane due to the presence of *Trichocephalus dispar*. Having had his attention drawn to blood in the dejecta in the course of an attack of beriberi (a phenomenon which has not been seen by other observers except on the very rarest occasions),¹ he found on post-mortem examination of the congested mucous membrane of the lower end of the ileum and of the cæcum, small defects

¹ Among a very large number of patients with beriberi, Scheube has found it in only two cases, both of them the rapid and malignant form of the disease. Simmons has found it only as a dysenteric complication. Other observers do not mention it at all.

of substance,¹ and in the intestinal canal large numbers of *Trichocephalus dispar*, some of which, he thought, had bored into the mucous membrane and caused those breaches of the surface. Besides that parasite he found a species of small round worm about 4 mm. long, his description of which is very obscure. Erni is of opinion that the nervous symptoms in the course of the malady are reflex phenomena, due to irritation of the intestinal mucous membrane, and that the complications of anæmia and dropsy are to be explained by the loss of blood caused by the parasites (as in the cachexia aquosa of *Anchylostoma duodenale*). Having these statements in view, Stammeshaus paid particular attention to the condition of the intestine in the cases of beriberi which came under his notice in the deadhouse. With a single exception he found *Anchylostoma duodenale* in the small intestine in all the cases (forty-eight in number), but mostly in very small numbers and far apart; but he found these parasites also in a fatal case of tropical dysentery, and in a case of cancer of the uterus. On the other hand, he never found *Trichocephalus dispar*. He concludes from his observations that *Anchylostoma duodenale* is very widely diffused in the Dutch East Indies; that that parasite is not the proper cause of beriberi; although it is not to be denied that it may have some influence in producing the characteristic anæmia. Wucherer and da Silva Lima have also found in the intestine, "small worms resembling *Anchylostoma duodenale*" in the bodies of several persons who had died of beriberi in Brazil.

¹ Wernich, who obtained only one post-mortem examination, found "extreme hyperæmia in the ileum, and at the lower end of it dark spots like hæmorrhages, which also occurred in the cæcum; in the colon the vessels were a good deal injected, but nothing else remarkable." Anderson, who had likewise only one post-mortem examination, found submucous ecchymoses at a few places in the upper part of the small intestine. Scheube, who examined three cases after death, found in one of them "a cluster of punctiform hæmorrhages in the ileum just above the valve, of various sizes up to 3 cm. long; the mucous membrane of the small intestine more or less injected at various points, and here and there swollen or œdematous;" in the second case, "the intestinal mucosa more or less injected over most of its extent, punctiform hæmorrhages at a few spots." The third case was complicated with typhoid, so that no conclusion could be drawn from the changes in the intestine, which might have belonged in part to the one disease and in part to the other.

§ 211. LEADING INDICATIONS FOR THE ETIOLOGY—NO EVIDENCE OF COMMUNICABILITY.

It must be obvious that none of these hypotheses and observations, although I have thought it necessary to reproduce them here as fully as possible, have dispelled the obscurity in which the nature of the morbid cause is shrouded, or have brought us any nearer to a solution of the problem ; and it is a task reserved for the future to continue the search after the specific element of the disease in one or other of the directions indicated. In this inquiry there are, it seems to me, four things to be specially kept in mind and attended to : firstly, the comparatively rare occurrence of the disease in childhood, in the female sex, and among the European part of the population ; secondly, the unusual frequency of cases among the crews of ships, or in bodies of troops quartered in localities where there is little or nothing of beriberi among the ordinary population ; thirdly, the rapid recovery of the patient on leaving the focus of disease, or on changing his mode of living ; and fourthly, the epidemics on board ship. That the true cause in these last cases, and others like them of which particulars have been given above (p. 588), is not merely an affair of “ mephitism ” developed from the crowding together of a number of persons in a small and ill-ventilated space, is shown by the fact that the same hardships occur every day on board transports and ships-of-war without beriberi developing, as well as by innumerable other instances of the same kind in all parts of the world.

If there should be any desire to speak of the unknown morbid agent under the name of “ miasma,” there can be no objection so long as nothing more is implied in the term than an unknown factor, and no prejudice entertained in favour of its telluric origin. At the same time it appears to me to be doubtful at the present date of writing (that is to say, in view of the observations published since 1860), whether the disease is to be reckoned among the infective diseases properly so called ; and I hold it to be still less justifiable, in regard to the etiology, to give way precipitately to the modern craze for bacteria in this particular field of inquiry, or to seek to establish the *communicability of the*

disease on the ground of casual and ambiguous facts. From India and the Malay Archipelago we have not a single fact affording certain proof that the disease is transmissible ; while against the idea, there is the fact that beriberi continues to be endemically prevalent in India at the present day within the same narrow limits to which it was confined at the beginning of the century. When it has broken out on board ship among Indian coolies, it has never spread to the attendants of the sick, the sailors and others ; and the observations of practitioners in Brazil are just as decidedly opposed to the importation or transmission of it by means of individuals or by goods.¹

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CHAPTER XVII.

SCROFULA.

§ 212. HISTORICAL REFERENCES TO SCROFULA.

The word "scrofula" or "scrophula" denoting an inflammatory kind of tumour, more particularly in the neck but also in other superficial parts of the body where there are many lymph-glands, such as the armpits and groins, is first met with in the medical writings of the school of Salerno.¹

¹ Scrofula or scrophula is derived from *scrofa* or *scrophæa*, a pig. It may seem to correspond etymologically to the word *χοιράς*, as derived from *χοῖρος*, a sucking pig; at all events Leonides says (in Aëtius, lib. xv, cap. v, ed. Basil., 1535, iii, 72): "Choerades nomine a suis mutuato appellantur: circa suum enim mandibulas adenosæ quidam globuli inveniuntur, quibus strumæ (choerades) assimilantur; sunt qui a copioso animalis partu nomen sumpsisse putent, quoniam et copiosa eorum propago est." Whether this derivation of the word *χοιράς* is the right one, or whether it should not rather be taken in a figurative sense (*χοιράς* meaning primarily "a stone"), appears to me to be open to question. Leonides is no trustworthy guide, inasmuch as he lived 600 or 700 years after the Hippocratic period, when the word first became a technical term. The latter explanation is certainly more promising than the former; and it may be supported by statements made in several passages of the writings of Galen ('Lib. de tumoribus præter naturam,' cap. xv, ed. Kühn, vii, 729, and 'Method. med.,' lib. xiii, cap. v, ed. cit., x, 881) to the effect that the inflamed glands are named *χοιράδες* when they assume a scirrhus hardness (*σκιρρῶθεντων ὄνομα*). In constructing the term "scrofula" the school of Salerno followed the interpretation of Leonides; the first of them to use the word was Constantinus Africanus, who employs it in several passages of his writings ('De morbis cognose. et curand.,' lib. vii, cap. xxiii, Opp. Basil., 1536, where the expression is "scrophulæ sunt dura apostemata in molli carne nascentia," and 'Lib. de chirurg.,' cap. xv, ed. cit., p. 333). After him we find the term used by Petrocelli, 'Practica,' lib. ii (in De Renzi's 'Collect. Salernitana,' iv, 287); then in the 'Tractatus de ægriitudinum curatione' (ib. ii, 461); and by Rolando, 'Chirurgia,' lib. ii, cap. xii (in Abulcasim's edition, Basil., 1541, p. 269), and in the 'Glossulæ quatuor magistrorum super chirurgiam Rogerii et Rolandi,' lib. ii, cap. v (in De Renzi's Coll., ii, 593), where the explanation is: "unde dicit scrophula a scrofa, quoniam sicut scrofa parit

It corresponds in meaning to the *χοιράς* of the Greeks, and to the "struma" of the Latin writers of antiquity, and, with some restrictions to our notion of scrofulous glandular tumours. As early as the Hippocratic collection there are indications pointing to the particularly common occurrence of these tumours in children, from which it may be inferred that the disease of *χοιράς* was for the most part the same as that which we have here to consider. The references are still clearer in the writings of the later Greek physicians, who associate the tumours with the glands in the neck, axilla and groin, characterising them as inflammatory swellings of the latter and dwelling upon the sluggish nature and protracted course of the inflammation.

In one passage of the Hippocratic writings¹ we read: "*πρεσβυτέροισι δὲ γενομένοισι* (i.e. in the somewhat older class of children) *χοιράδες καὶ τὰλλα φύματα μάλιστα δὲ τὰ προειρημένα.*" and in another²: "*περὶ δὲ ἡλικιών, φύματα μὲν ἔμπυα καὶ τὰ χοιρώδεα, ταῦτα πλείστα τὰ παῖδια ἰσχοῦσι καὶ ῥᾶστα ἐξ αὐτέων ἀπαλλάσσει· τυῖσι δὲ γεραιτέροισί τε τῶν παιδίων καὶ νεηνίσκοισι φύεται μὲν ἱλάσσω, χαλεπώτερον δὲ ἐξ αὐτέων ἀπαλλάσσουνσι.*" In the post-Hippocratic treatise 'De glandulis,' it is stated in regard to the inflammation of the cervical glands:³ "*καὶ ἡ φλεγμονή, στάσιμον ὄν ὑγρὸν, χοιράδες ἐγγίνονται.*" Galen in several passages⁴ says that, if the inflamed glands (*βουβῶνες*) take on a scirrhus character, or become hard, they are then known by the name of *χοιράδες* (*σκιρρωθέντων ὄνομα*), and that these are not prone to suppurate (*εἰς ἐμπύησιν*) inasmuch as they proceed, not *ὑπὸ θερμῆς ὕλης* (ex calida materia), but from "a mucous and cold dyscrasia."⁵ Leonides⁶ writes to the same effect:

multiplies fetus, ita hujusmodi passio generat semper multas scrophulas." In the medical writers of the latter part of the middle ages, such as Gordon ('Lilium medicum,' De apostematibus frigidis., Practic. i, rubr. 2, Lugd., 1574, p. 90), John of Gaddesden ('Rosa Anglica,' Aug. Vindel., 1595, p. 981), Guido ('Chirurg. Tract.,' ii, cap. iv, Lugd., 1572, p. 77), and Valescus de Tharanta ('Philonium,' lib. vii, cap. 29-30, Lugd., 1490, fol. 337 b), we continue to meet with the term "scrophula." It is not until the sixteenth century that it begins to give place more and more to "struma," which now got to be used by medical writers indiscriminately for scrofula and goitre, and continued to have this double significance until the eighteenth century, when scrofulous disease was thoroughly investigated, and the old name was revived to distinguish it from goitre. It is only in England that the adjective "strumous" is still applied to cases of scrofula. The term "scrofulosis," to signify the process underlying the disease, is of recent date.

¹ 'Aphorism.,' sect. iii, § 26, ed. Littré, iv, 498.

² 'Prædictor.,' lib. ii, § 11, ed. cit., ix, 30.

³ § 2 and 7, ed. c. viii, 556, 562.

⁴ Ll. cc. and 'De locis affectis,' lib. i, cap. iii, ed. c. viii, 31.

⁵ 'Comment. in Hipp. Aphor.,' cap. xxxvi, ed. c. xvii, B. 637.

⁶ L. c.

"Sunt autem strumæ (*i.e.* χοιράδες) carnes subcandidæ auctu faciles, intra membranam contentæ, utque summatim dicam glandulæ induratae, unde et in collo, axillis, inguinibus fere generantur, ubi glandulæ vasis substratæ collocantur."

The same conception and description of the form of disease is met with in the works of such of the later Greek and Roman¹ writers on medicine as treat of the subject at all, including Paulus Ægineta² and Theophanes Nonnus;³ also in those of the Arabian school,⁴ the school of Salerno,⁵ and in other physicians of the middle ages.⁶ And although the diagnosis of the tumours in question in all these authorities is by no means a very precise one, although they have plainly confounded with them various other forms of tumour (particularly syphilitic glandular swellings) not only in the naming but also in fact; it is none the less certain from their accounts that glandular swellings played the chief part among those χοιράδες, "scrofulæ" and "strumæ"; and we may find evidence that even the specific character of the disease was to some extent correctly recognised, in the references to its especial frequency in childhood, to the "cold and mucous nature" of the tumours, and to the special features in the course of the malady dependent thereon, whereby it was distinguished from "phlegmonous" inflammations.

This acquaintance with the scrofulous disease of external lymph-glands, and discrimination of them from other glandular tumours, came out still more definitely in the medical writings of the sixteenth and seventeenth centuries. But it was not until the close of the seventeenth century that the intimate connexion between the glandular affection and

¹ Celsus, lib. v, cap. xxviii, § 7, ed. Almeloveen. Basel, 1748, 323: "Struma est tumor, in quo subter concreta quaedam ex pure et sanguine quasi glandulae oriuntur."

² Lib. vi, cap. xxxv, Lugd., 1551, 385.

³ Epitome, cap. cxxiv, ed. Bernard, Gotha, 1794, i, 378.

⁴ See Rhazes, 'De re medica,' lib. vii, cap. viii, and 'Divisionum,' lib. i, cap. cxxix, in Opuscula, Basil., 1544, 167, 427; also Abulcasim, 'De Chirurgia,' lib. i, cap. xii, and lib. ii, cap. xlii, ed. Channing, Oxon., 1778, 51, 223, ed. Leclerc, Par., 1861, 29, 118; and Avicenna, 'Canon,' lib. iv, fen. iii, Tract. ii, cap. ix, ed. Venet., 1564, ii, 123.

⁵ See the authors quoted in note 1.

⁶ Conf. *ibid.* and Actuarius, 'Method. med.,' lib. ii, cap. xii, in Stephani, 'Collect.,' 190.

other local lesions, either coexistent or subsequent, began to be kept more directly in view ; and thus there arose the notion of "scrofulous disease" as the outcome of constitutional errors of nutrition depending on a morbid diathesis. Next to the works of Wiseman,¹ Cullen,² and other British authorities, the writings that contributed most to instruct the great medical public on this subject were the articles³ by Faure, Bordeu, Charmetton, Majault and Goursaud, which were sent in as prize essays to the Académie de Chirurgie of Paris and were published by it ; also the writings of Hufeland⁴ and Weber⁵ connecting with these ; but most of all the excellent work of Kortum.⁶ However, it was not long before writers began to indulge in vague speculations on the nature of the disease, resorting for an explanation of its pathogenesis to the idea of a "scrofulous acrimony." At the same time the scrofulous disease became the Alpha and Omega of practice in children's diseases ; and for that period of life it very soon came to take the place that the "hæmorrhoidal disease," inaugurated by the Stahlian doctrine of the golden vessel, had won for itself in the ill health of later life. As Henle⁷ says, scrofula was a bugbear that was made, with no obvious or sufficient reason, to carry everything of a morbid kind that befel a child under fourteen years of age. Pathological anatomy put an end to that unreal state of things ; but it conducted to an opposite extreme. It taught that there was nothing specific in the whole series of morbid changes in scrofula, that it was a matter of chronic inflammatory processes in various tissues,—lymph-glands, mucous membranes, the skin and the bones,—such as were found in many other diseases ; and it looked, to quote Henle again, as if the ancient family of scrofula were about to perish through their own excesses. In this movement, indeed, the good was
rid of with the bad. Those who looked at things from

1 'Eight chirurgial treatises.' Lond., 1696, Nr. iv : 'Of the King's evil.'

2 'First Lines in the Practice of Physic,' Ger. ed., Leipz., 1785, iv, 190.

3 'Recueil des pièces qui ont concouru pour le prix de l'Acad. roy. de Chirurgie,' Paris, 1759, iii, 21—351.

4 'Ueber die Natur . . . der Scrofelkrankheit,' Berl., 1785.

5 'Von den Scropheln u. s. w.' (einz.) Theil, Salzburg, 1793.

6 'Commentarius de vitio scrofuloso,' ii tomi, Lemgo, 1789, 90.

7 'Handbuch der rationellen Pathologie,' Braunschweig, 1847, ii, 376.

the gross anatomical point of view, took no account of clinical facts; they made the mistake of thinking that the peculiarities of a disease were really to be sought in the grouping of the local lesions, that the anatomical analysis of all diseases whatsoever conducts us to a comparatively small number of elementary pathological processes, and that it is the way in which these are grouped together that determines those peculiarities of each concrete disease which we judge of from the clinical point of view. That was another one-sided doctrine which has been overthrown in its turn, and that too without any falling back into the old error; so that at the present day it is not seriously disputed that the scrofulous morbid process, comprising the various local lesions of which it is made up, is marked by a unity and specificity such as secure for it, as scrofulosis, a place among the constitutional disorders of nutrition. Or, if the conclusion that has been drawn from Koch's discovery of so-called tubercle bacilli in scrofulous glands,—that the parasite is the specific excitant of the disease,—be a genuine one, its place would be among the parasitic diseases.

Although this brief summary of the development of the doctrine of scrofula does not, strictly speaking, belong to my proper subject; yet it seemed to me desirable to insert it in order to show what difficulties are encountered in the *history of scrofulosis*, difficulties in answering the questions as to the changes in the amount of the disease in the course of centuries and as to any differences noticeable within the several periods, and whether scrofula, as often asserted, has undergone any considerable increase of late. These difficulties arise from the circumstance above mentioned that the notion of "scrofula" was until not very long ago an altogether indefinite one; that until the sixteenth century, and even later, various forms of glandular swellings and other sorts of tumours about the neck, even goitre itself, were received in the group of *χοιράδες*, "scrofulæ" or "strumæ," while we are at this distance of time unable to judge from the descriptions of observers how much belonged to one thing and how much to another; and that, in the last century and early part of the present, after the process of scrofula had become familiar in

its various pathological expressions, every conceivable form of children's disease which men did not know what else to make of was brought under scrofula, so that scrofulosis dominated the whole of pædiatrics.

Regarding the history of scrofula, all that one can say with safety is that the malady has been prevalent at all times. On the other hand, it is a question whether any considerable increase in its amount has taken place in recent times, or whether the increase is not rather an apparent one, depending on an erroneous extension of the meaning of the term.¹ But it is a quite unproven assumption, and I am convinced that it is an altogether erroneous one also, that scrofula did not attain its present wide diffusion in Europe until the inoculation with the small-pox and vaccination were introduced, and until the potato became a general article of diet. This is a clear instance of the clumsy fallacy; two events coincide in time, namely, the introduction of those beneficent innovations, and the retrograde enlargement of the idea of "scrofula," and these have been brought into connexion; or, in other words, there has been first of all a mystification as to terms, and on the top of that the fallacy of *post hoc ergo propter hoc*. At the same time it is not to be denied that the malady has undergone ups and downs in the course of centuries at various parts of the world, according as the etiological factors favouring its production have been to the fore or not; and, as we shall see in the sequel, it is only within the very latest times that the disease has become at all general in some countries.

¹ On the other hand, Phillips ('Scrofula: its Nature, Causes, &c.,' Lond., 1846, p. 92), deduces from the mortality statistics the conclusion that the disease in London had declined considerably between 1700 to 1831. While the mortality from scrofula in 1700 was in the proportion of 1 to 9'80, it amounted in 1831 only to 1 in 135'89. It is clear that this statistical result possesses no value; in the first place it is derived from a small number of cases (in 1700 there were 73 deaths returned from scrofula in a population of 665,000, and in 1831 there were 9 deaths in 1,233,000); and in the second place the materials on which the statistics are based do not deserve to be trusted in the slightest degree.

§ 213. PRESENT GEOGRAPHICAL DISTRIBUTION.

Scrofula has very decidedly the character of an ubiquitous disease; its *geographical area* extends over the whole of the habitable globe. In some regions and in particular tracts of country it is seen more frequently than in others; although we are not warranted in saying that it is anywhere properly *endemic*. No definite or numerically expressed measure of the number of cases in the several regions is attainable; for the statistics are either wanting, or, in the case of the statistics of hospitals and the mortality returns, they are for obvious reasons unsuited to such a purpose. The most useful tables in this respect are the recruiting lists, giving the number of recruits rejected as unfit for service on account of scrofula; although even these have a limited value, as affecting only a somewhat mature age and only the male sex. Accordingly in the account that follows of the frequency of the malady, I must content myself with repeating the general terms in which the authorities speak of the number of cases, such terms as "very common," "common," "rare," and the like.

Europe is truly classic ground for scrofula; for there is scarcely a single considerable tract of country in it that is not affected more or less. In the *Iberian Peninsula*,¹ the head quarters of the malady are the large towns, both those on the elevated table-land of the interior and those on the plains and the sea coast; and even places with a specially good climate, such as Valencia and Cadiz, are not free from it. In Lisbon scrofula is so prevalent that in 1842, according to the returns of Rozas,² the orphanage of that city designed for children from four to sixteen years of age, contained 279 among its 800 inmates (or 35 per cent.), who showed the most undoubted signs of scrofula. The diffusion of the disease in *Italy* may be gathered from the figures

¹ Faure, 'Souvenirs du midi, etc.' Lugol, 'Untersuchungen und Beobachtungen über die Ursachen der scrophulösen Krankheiten,' From the French. Leipz., 1845, 214; Phillips, l. c., 87; Trogher, 'Briefe während einer Reise durch Istrien u. s. w.,' Triest, 1855, 137, 157, 161.

² Phillips, 319.

extracted by Sormani¹ from the conscription lists of 1863-1876. According to these returns, the number of persons of the age of twenty who were rejected for scrofula amounted to 3·5 per 1000 of those examined. According to provinces and circondarias they were distributed as follows :

Valsesia (Piedmont)	. . 10·8	Pistoja (Tuscany)	. . 6·3
Malfi (Basilicata)	. . 9·5	Massa (Tuscany)	. . 6·2
Domodossola (Piedmont)	. . 8·8	Mazzara (Sicily)	. . 6·1
Parma (Æmilia)	. . 8·8	Udine (Venetia)	. . 5·6
Milan (Lombardy)	. . 7·8	Civita Vecchia (Rome)	. . 5·6
Paola (Calabria)	. . 7·7	Sondrio (Lombardy)	. . 5·4
Porto Maurizio (Liguria)	. . 7·6	Perugia (Umbria)	. . 5·0
Pavia (Lombardy)	. . 7·5	Orvieto (Umbria)	. . 5·0
Mantua (Lombardy)	. . 7·1	Rieti (Umbria)	. . 5·0
Monza (Lombardy)	. . 6·7	Brescia (Lombardy)	. . 4·7
Pisa (Tuscany)	. . 6·7	Fermo (Marches)	. . 4·1
Vergato (Æmilia)	. . 6·6	Rovigo (Venetia)	. . 2·8
Como (Lombardy)	. . 6·5		

From the results of this inquiry, which are borne out and in part amplified by the special accounts referred to in the notes, it follows that scrofula is very common all over Italy ; as Parola² says, “fra le cachessie la più diffusa.” But its distribution throughout the country is very unequal, and these inequalities are altogether independent of the geographical position of the several places or of their elevation above the sea-level. The worst districts are some of those in Piedmont, especially the circondarias of Aosta, Cuneo, Vercelli and Novara ;³ in Lombardy⁴ the circles of Milan, Pavia, Mantua, Como, Sondrio, Bergamo, Cremona ; in Venetia,⁵ particularly the provinces of Venice, Vicenza and Udine, while Verona enjoys a remarkable immunity ;⁶ in

¹ ‘Geografia nosol. dell’ Italia,’ Roma, 1881, 143.

² ‘Saggio di climatologia e di geogr. nosol. dell’ Italia,’ Torino, 1881, 494.

³ Dubini, ‘Gaz. med. di Milano,’ 1847, Nr. 46; Maffoni, ‘Atti dell’ Acad. med.-chir. di Torino,’ ii, 453.

⁴ See references in ‘Oest. med. Jahrb. Neueste Folge,’ xi, 19, xxi, 3; Hildenbrand, ‘Annal. schol. clin. Ticin,’ Papiæ, 1826, i, 117; Speranza, ‘Annal. univ. di med.,’ 1856, Marzo, 449; Comolli, ‘Gaz. med. di Milano,’ 1848, 305; Tassani, ib., 1847, 173; Balardini, ‘Topogr. stat. med. della provincia di Sondrio,’ Milano, 1834, 55.

⁵ Taussig, ‘Venedig und seine klimat. Verhältnisse,’ Venedig, 1847; Parola, l c.

⁶ Agostini, ‘Annali, univ. di med.,’ 1874, Dcbr., 478; Parola, l c.

Liguria, the circondarias of Genoa, and Porto Maurizio;¹ in Tuscany, the districts of Pisa, Pistoja, Massa and Siena;² in Roman territory, Civita Vecchia;³ in the Æmilia, the towns of Ferrara and Bologna;⁴ in the Marches, Ancona;⁵ in Campania, the circondarias of Naples and Terra di Lavoro;⁶ in Apulia, Foggia;⁷ and in Sicily, Palermo. It is common also in Sardinia,⁸ but in Corsica it would appear to be surprisingly rare.⁹

For *France* also there are statistical returns¹⁰ of the amount of scrofula in the recruiting lists from 1831 to 1853. According to these the malady occurred at the rate of 10 per 1000 of those examined, or three times as often as in Italy.¹¹ This average sick-rate was distributed among the several departments as follows

In 1 Dpt. (Pas de Calais)	.	.	1'2	per 1000
„ 3 Dpts. (Pyrén. orient., Gironde, Vendée)	.	.	4'0—5'0	„
„ 6 „ (Basses-Alpes, Gers, Indre, Charente, Eure, Morbihan)	.	.	5'2—6'0	„
„ 11 „ (Hérault, Indre-Loire, Haute-Garonne, Hautes-Pyrén., Somme, Haute-Vienne, Vaucluse, Tarn-Garonne, Seine-Marne, Meurthe, Doubs)	.	.	6'2—7'0	„
„ 12 „ (Ille-Villaine, Gard, Lot-Garonne, Seine-Oise, Aude, Haute-Marne, Ardennes, Corrèze, Seine infér., Calvados, Haute-Saône, Bouches-du-Rhône)	.	.	7'1—8'0	„

¹ Speranza, l. c.; 'Descrizione di Genova,' &c., 1846.

² Speranza, l. c.; Danesi, 'Relaz. topogr. . . della città di Siena,' &c., Siena, 1842.

³ Jacquot, 'Gaz. méd. de Paris,' 1853, 532.

⁴ Parola, l. c.

⁵ Briard, 'Travaux de la Soc. de méd. de Dijon,' Ann., 1834—37, 122.

⁶ de Renzi, 'Topogr. statistica-medica della città di Napoli,' &c., Nap., 1845; Parola, l. c.

⁷ *Ib.*

⁸ Moris in de la Marmora, 'Voyage en Sardaigne.'

⁹ Vanucci, 'Bull. de l'Acad. de méd.,' 1838, Mai.

¹⁰ According to Boudin, 'Traité de géographie et statist. méd.,' Par., 1857, ii.

¹¹ According to Chervier's figures ('Annal. de démographie,' 1880), taken from the conscription lists of the French army for the years 1850-69, the mean rate of sickness from scrofula was 17'04 per 1000. The enormous difference in the amount of the disease between the male populations of Italy and of France is no doubt explicable in great part by the fact that the French military surgeons took a much wider view of the term "scrofula" than the Italian; but it may be explained in part also by the greater prevalence of the disease in France.

In 15 Dpts.	(Sarthe, Jura, Cher, Loire infér., Côtes-du-Nord, Mayenne, Meuse, Ardèche, Charente infér., Marne, Côte-d'Or, Tarn, Maine-Loire, Ain, Finistère).	8'1—9'0	per 1000
„ 16 „	(Yonne, Eure-Loir, Lot, Vienne, Drôme, Var, Loire-Cher, Basses-Pyrén., Manche, Arriège, Allier, Isère, Creuse, Bas-Rhin, Dordogne, Saône-Loire)	9'1—10'0	„
„ 8 „	(Moselle, Seine, Puy-de-Dôme, Aisne, Orne, Aube, Aveyron, Hautes-Alpes)	11'0—12'0	„
„ 8 „	(Loiret, Vosges, Haut-Rhin, Rhône, Landes, Deux-Sèvres, Loire, Oise)	13'0—18'0	„
„ 5 „	(Haute-Loire, Lozère, Cantal, Nord, Nièvre)	20'0—30'0	„

These facts, taken along with more special information, teach us that in France there are a few great centres of scrofula which do not owe their pre-eminence, we may remark at the outset, to its prevalence in large towns such as Havre, Lille, Nantes, Paris, Rheims, Strasburg, Toulouse, and Marseilles, or at all events not to the urban element alone. One of the greatest of these centres includes the south-eastern departments of Hautes-Alpes, Isère, Rhône, Loire, Haute-Loire, Lozère, Cantal and Aveyron (comprising a large part of Dauphiné,¹ Lyonnais² and Languedoc); and in that region the average sick-rate from scrofula is 15 to 20 per 1000. Joining on to that is another area, including the departments of Saône-Loire, Allier, Puy-de-Dôme, Creuze, Nièvre and Loiret, and extending from Auvergne through Bourbonnais and Nivernais;³ also a focus in the Jura (Franche Comté),⁴ another in Alsace (Haut-Rhin, Bas-Rhin and Vosges),⁵ and a third in the north of the country, par-

¹ See Grange, 'Annal. de Chimie et de Phys.,' xxiv, 364; Lepelletier, 'Traité complet sur la maladie scrofuleuse,' Paris, 1830.

² Maring et Quesnois ('Topogr. et stat. méd. du Dpt. du Rhône,' Lyon, 1866), estimate from the conscription lists from 1854 to 1863 that the number of persons rejected as unfit for military service on account of scrofula in the Department of the Rhône was 13'8 per 1000, and that in some cantons the figures rose to 22'5, and even to 30 per 1000.

³ See Brieu de, 'Hist. et mém. de la soc. roy. de méd.,' v, Mém., 306 (referring to Upper Auvergne).

⁴ Germain, 'Annal. d'hyg.,' 1850, Juli, 123.

⁵ Didelot, 'Hist. et mém. de la soc. roy. de méd.,' ii, Hist. 135; Cuynat, Travaux de la soc. de med. de Dijon,' 1832, 22; Georgeon, 'Considér. gén. sur

ticularly in the Department du Nord, where the colliery districts, as well as the city of Lille, furnish a very considerable contingent of the cases.¹

In *Switzerland* scrofula is prevalent in the large towns, and, after them, more especially in the deep-cleft valleys on the slopes of the Jura and the Alps; most of all in the Rhone valley from Vallais to St. Maurice, next in the Canton of Bern and in the valleys running towards Lake Lucerne, and to a less extent among the High Alps and on the plains.²

In *Belgium*, as all the authorities agree, it is only since the first quarter of this century that scrofula has come to be so generally diffused³ as we now find it, particularly in East⁴ and West⁵ Flanders and in Antwerp.⁶ *Holland*, on the other hand, has been from the first and is still one of the chief seats of scrofula.⁷

Next we come upon a very extensive region of scrofula in *Germany* and *Austria*. Besides the large towns such as Munich,⁸ Vienna, Stuttgart,⁹ Dresden,¹⁰ Leipzig,¹¹ Berlin,¹² Stettin,¹³ Hamburg, Danzig and Breslau,¹⁴ there are many

P'hygiène dans les campagnes de la partie montagneuse des Vosges,' Strasb., 1863, 27.

¹ Bouisson, 'Étude méd. sur l'ouvrier houillier,' Par., 1866.

² See Lebert, 'Lehrbuch der Scrophel- und Tuberkelkrankheiten.' From the French. Stuttg., 1851, 46; and Lombard, 'Traité de climatol. méd.'

³ Meyne, 'Topogr. méd. de la Belgique,' Brux., 1865, 116.

⁴ Overloop, 'Annal. de la Soc. de méd. de Gand,' 1842, Oct.; Waldeck, ib., 1845, Jan., 69.

⁵ Woets, 'Annal de la Soc. de méd. de Bruges,' i, 17.

⁶ Thys, 'Annal. de la Soc. de méd. d'Anvers,' 1845, 37; Luyks, 'Arch de la méd. belge,' 1845, Juin, 78; Peutermans, ib., Aug., 181.

⁷ Thyssen, 'Geschiedk. beschouw der ziekten in de Nederlanden,' Amsterd., 1824; Dolleman, 'Disquis. hist. de plerisque apud Belgas septentr. endemiis morbis,' Amstel., 1824, 65; Guislain, 'Annal. de la Soc. de méd. de Gand,' 1842, Jan.

⁸ According to information obtained by Phillips, two thirds of all the children received into the Munich orphanage suffer from scrofula.

⁹ Plieninger, 'Beschreibung von Stuttgart u. s. w.,' Stuttg., 1834.

¹⁰ Meyer, 'Versuch einer med. Topographie . . . von Dresden,' Stolberg, 1840, 253.

¹¹ Krug, 'Acta policlinica,' Lips., 1841, 60.

¹² The scrofulous children among the inmates of the Friedrich Orphanage at Berlin are 53 per cent. of the whole.

¹³ Müller in 'Hufeland's Journ. der Arzneikde,' 1843, Juni, 90.

¹⁴ Graetzer, 'Beitr. zur med. Statistik der Stadt Breslau,' Bresl., 1834.

other foci of it of greater or less extent, such as the Dittmarschen,¹ the Harz² country, the Saxon Metal Mountains,³ Upper Silesia,⁴ the valleys of the Giant Mountains,⁵ many parts of Westphalia,⁶ Thuringia,⁷ Odenwald,⁸ certain mountainous circles of Bohemia,⁹ several parts of Upper Austria,¹⁰ Salzburg,¹¹ Styria,¹² and the Austrian Military Frontier.¹³

In *Great Britain* the populous centres of trade and manufacture have always been the principal seats of scrofula.¹⁴ Phillips estimates from an enumeration of sick persons (which is, however, a very untrustworthy one) that an average of 24·5 per cent of the population in England are affected with scrofula, the rate falling in some parts of the country to 11 per cent. and in others rising to the (extremely questionable) figure of 72 per cent. In *Ireland* also, according to Wylde's¹⁵ researches, the malady has been prevalent in widest diffusion from the earliest times. In the *Shetland Islands* scrofula is said to be indigenous in almost every family.¹⁶

¹ Dohrn in 'Pfaff's Mittheilungen,' Neue Folge i, Heft 6, 32.

² Klinge in 'Hufeland's Journ. der Arzeneikde.,' 1798, vi, 902; Wendelstadt, ib., 1801; xii, Heft 2, 125; Fuchs, 'Hannov. Annal. der Heilkde,' 1840, v, 73.

³ Petrenz, 'Wöchentl. Beitr. zur Klinik, 1833, i, 245; Ettmüller, ib., 1834, i, 611.

⁴ Lorinser, 'Pr. med. Vereins-Ztg., 1833, No. 12.

⁵ Preiss, 'Die klimatischen Verhältnisse des Warmbrunner Thales u. s. w.,' Bresl., 1843.

⁶ Nicolai in 'Rust's Magazin,' xxxix, 97; 'Sanitätsberichte aus Westfalen,' 1845, 45.

⁷ Fuchs, 'Topogr. des Kreises Schmalkalden,' Marb., 1848; Lübben, 'Correspondenzbl. des ärztl. Vereins von Thüringen,' 1880, Nr. 4, 112. According to the reports of Thuringian practitioners for 1874 and 1875, scrofula (and rickets) were found most seldom in the valley of the Werra, next to that in the mountainous districts, and most commonly in the Thuringian basin at elevations of 150 to 300 metres (500 to 1000 feet).

⁸ Ebel in 'Hufeland's Journ. der Arzeneikde.,' 1840, Juni, 106.

⁹ 'Oest. med. Jahrb.,' 1840, Nste. Folge, xxiv, 608, 1843, ii, 354, 1845, iv, 234.

¹⁰ Ib., 1831, Nste. Folge, i, Heft 4, 46, 1834, vii, 359, 1840, xxiv, 265.

¹¹ Ib., 1836, xi, 391, 1844, iv, 360; Maffei, 'Der Cretinismus u. s. w.,' 175.

¹² Pilz, 'Oest. med. Jahrb.,' 1848, i, 357, iii, 80; Macher, 'Med.-statist. Topographie des Herzogthums Steyermark,' Graz, 1860.

¹³ Müller, 'Oest. med. Jahrb.,' 1842, i, 227, 340, 1843, iv, 343.

¹⁴ See Autenrieth, 'Uebersicht der Volkskrankheiten in Grossbritannien,' Tübing, 1823, 93; Forbes, 'Transact of the Prov. Med. Assoc.,' iv, 189; Alison, 'Lancet,' 1841-42, i, 800.

¹⁵ 'Edinb. Med. and Surg. Journ.,' 1845, July 11, 12, 16.

¹⁶ Sexby, in 'Dobell's Reports,' 1871, 522.

Accounts the same as these of the common, or, as it is called, endemic occurrence of scrofula, come to us from *Denmark*,¹ *Norway* and *Sweden*. In *Sweden* (as in *Belgium*) it is only since the beginning of the century, according to *Huss*,² that the malady has appeared in some districts, such as *Angermanland*, and in others attained its present general diffusion. The worst districts are *Malmöhus*, *Halland*, *Calmar*, *Jönköping*, *Skaraborg*, *Bohus*, *Nyköping*, *Upsala*, *Stockholm* and *Fahlu*.³ The assertion of *Schleisner*⁴ that scrofula is rare in *Iceland* is contradicted by *Finsen*.⁵ But *Manicus*⁶ and *Panum*⁷ agree that it is rare in the *Faröe Islands*, being seen, according to the latter authority, only in the children of Danish families.

There are many accounts of the occurrence of scrofula throughout the enormous extent of the *Russian Empire*; and these, casual though they be, enable us to conclude that the cases are numerous and widely distributed. This applies most to *Poland*,⁸ *St. Petersburg*,⁹ and the *Baltic Provinces*;¹⁰ but there are corresponding accounts from *Kovno*,¹¹ *Mohilev*,¹² *Jaroslav*,¹³ *Novgorod*,¹⁴ *Kursk*,¹⁵ *Kasan*,¹⁶ *Viatka*,¹⁷ *Kishniev*,¹⁸

¹ Otto, in 'Rust's Magaz. für Heilkde,' liv, 203.

² 'Om Sverges endemiska Sjukdomar,' Stockh., 1852, 9.

³ Huss, l. c., 18, 20, 54, 67, 87; and Berg, 'Bidrag till Sveriges med. Topografi och Statistik,' Stockh., 1853, a. v. O.

⁴ 'Island undersögt, &c.,' 3.

⁵ 'Jagttagelser angående Sygdomsforholdene i Island,' Kjöbenh., 1874, 57.

⁶ 'Bibl. for Laeger,' 1824, i, 15.

⁷ Ib., 1847, i, 277, 310.

⁸ Theiner, 'Magazin für Heilkde. in Polen,' 1828, 224.

⁹ Attenhofer, 'Med. Topogr. der Hauptstadt St. Petersburg,' Zürich, 1817, 230; Heine, in 'Schmidt's Jahrbh.,' 1838, xvii, 224; Lichtenstadt, in 'Hecker's wissensch. Annal. der Heilkde.,' 1834, xxx, 76; Doepp, 'Verm. Abhandl. deutscher Aerzte in Petersburg,' 1835, v, 310. According to Phillips (l. c., 88) there were 343 children with scrofula among the 840 inmates of the Foundling Hospital.

¹⁰ Moritz, 'Specimen topogr. med. Dorpatensis,' Dorp., 1823.

¹¹ Weljamowitsch, 'Med. Ztg. Russl.,' 1848, 134.

¹² Kleinenberg, ib., 1847, 410.

¹³ Scholvin, ib., 1848, 331.

¹⁴ Bardowski, ib., 1850, 171.

¹⁵ Guttzeit, ib., 1851, 244.

¹⁶ Erdmann, 'Med. Topogr. des Gouvernem. Kasan,' &c., Riga, 1822, 159, 252; Blossfeld, 'Petersb. Journ. für Natur- und Heilkde.,' Nr. 4, 151.

¹⁷ Jonin, 'Med. Ztg. Russl.,' 1849, 45.

¹⁸ Heine, ib., 1845, 80.

Odessa,¹ Astrakhan,² and Orenburg.³ In the Crimea (Sebastopol) it would seem to be rare,⁴ and to be altogether unknown among the Kirghiz hordes.⁵ In the mountainous parts of *Transcaucasia*, such as Grusia, scrofula appears to be rare,⁶ but in other parts of that country it is often met with.⁷ From various parts of *Siberia* also, such as Tomsk,⁸ the region of Lake Baikal (among the Buriats),⁹ and Vladivostock,¹⁰ we have accounts of the frequent occurrence of scrofula. In *Hungary*¹¹ it would appear to be not less widely spread than in Russia. In *Roumania*¹² and *Montenegro*¹³ it takes a foremost place among the chronic diseases; and the same is true of *Turkey*,¹⁴ where it is met with in the larger communities not less frequently than in Paris and other European cities.¹⁵ The opinion of Wibmer¹⁶ that scrofula is rarer in *Greece* than in Western Europe is opposed by the statements of Kay¹⁷ and Pallas,¹⁸ who are agreed as to its great frequency; and to the same effect are the accounts of Hennen,¹⁹ Horner,²⁰ Ferrara,²¹ and others for the *Ionian Islands*.

In *Asiatic territory* scrofula is prevalent to the same

¹ Andrejewsky, in 'Graefe und Walther's Journal,' xx, 277.

² Herrmann, 'Med. Ztg. Russl.,' 1845, 187.

³ Maydell, 'Nonnulla topogr. med. Orenburg. spect. Dorpat,' 1849.

⁴ Heinrich, 'Med. Ztg. Russl.,' 1845, 380.

⁵ Maydell, l. c.

⁶ Ref. in 'Hecker's wissenschaftl. Annal.,' 1835, xxxi, 331.

⁷ Hirtzius, 'Russ. Samml. für Natur- und Heilkde.,' i, 561.

⁸ Rex, 'Med. Ztg. Russl.,' 1859, 408.

⁹ Haupt, ib., 1845, 376.

¹⁰ Sollaud, 'Arch. de méd. nav., 1882, Sptbr., 196.

¹¹ See Jankovich, 'Pesth und Ofen mit ihren Bewohnern u. s. w.,' Ofen, 1838, 304; Bartsch, 'Ungar. Zeitschr. für Natur- und Heilkde.,' iii, No. 30.

¹² Champouillon, 'Mém. de méd. milit.,' 1868, Mars, 191; Leconte, 'Considér. sur la pathol. des provinces du Bas-Danube,' Montp., 1869, 42.

¹³ Boulogne, 'Mém. de méd. milit.,' 1868, Debr., 486.

¹⁴ Oppenheim, 'Ueber den Zustand der Heilkde. . . in der Türkei,' Hamburg, 1833, 63; Rigler, 'Die Türkei u. s. w.,' ii, 416; Thirk, 'Oest. med. Wochenschr.,' 1846 781.

¹⁵ Beyran, 'Gaz. méd. de Paris,' 1854, 342.

¹⁶ In Schoepff, 'Jahresber. zur pract. Med.,' 1841.

¹⁷ In Phillips, 90.

¹⁸ 'Annali univ. di med.,' 1842, Aprile.

¹⁹ 'Sketches,' &c.

²⁰ 'Med. and topogr. observations upon the Mediterranean,' Philad., 1839.

²¹ 'Topogr. méd. de l'île de Leucade,' &c., 1827.

extent as in the countries of Europe just mentioned. It occurs on the coasts and in inland districts of *Syria*,¹ *Mesopotamia*² and *Arabia*³ (particularly in the southern highlands of Nejd). For other parts of Nearer Asia, such as *Persia* and *Turkestan*, I do not know of any accounts relating to scrofula. From India, on the other hand, we have numerous reports which go entirely against the assertions of Scott,⁴ Morehead,⁵ and others that scrofula is rare in that country, or of Ewart,⁶ that it is a good deal less common than in Europe. Gordon's observations in Bengal lead him to say that "scrofulous affections of the cervical as well as the mesenteric glands were the most frequent ailments among children of both sexes, and the mortality from the latter cause exceedingly great,"⁷ and the considerable frequency of the malady is in like manner asserted by Shortt,⁸ Huillet,⁹ Eyre,¹⁰ and Day,¹¹ for various parts of the Madras Presidency; by Annesley¹² for Mysore, Kinnis¹³ for the Bombay Presidency, Gibson¹⁴ for Gujerat, Hinder¹⁵ for Umritsur (Lahore), and McClelland¹⁶ for Kumaon.

An illustration of the commonness of the malady in India is afforded by the results of an enumeration of scrofulous children in the schools of Calcutta and other cities, which was made at the instigation of

¹ Pruner, 'Die Krank. des Orients,' Erlang., 1846, 321; Tobler, 'Beitr. zur med. Topogr. von Jerusalem,' Berl., 1855, 56; Barret, 'Arch. de méd. nav.,' 1878, Août, 87; Robertson ('Edinb. Med. and Surg. Journ.,' 1843, April, 247), observes that scrofula is rare in the mountainous districts of Syria.

² Floyd, 'Lancet,' 1843, Nr. 4.

³ Pruner, l. c.; Palgrave, 'l'Union méd.,' 1866, 308.

⁴ 'Journ. of Sc. and Arts.,' i, Nr. 2.

⁵ 'Clinical Researches,' &c.

⁶ 'Lancet,' 1881, May, 784.

⁷ 'Med. Times and Gaz.,' 1855, Decr., 538.

⁸ 'Madras Quart. Journ. of Med. Sc.,' 1866, July.

⁹ 'Arch. de méd. nav.,' 1868, Févr., 82.

¹⁰ 'Madras Quart. Journ. of Med. Sc.,' 1860, Octbr., 340. In Bellary, 1851-55, 564 cases of scrofula were admitted, or 7 per cent. of the whole number of patients.

¹¹ *Ib.*, 1862, Jan., 33.

¹² 'Researches into the More Prevalent Diseases of India,' Lond., 1841, 109.

¹³ 'Edinb. Med. and Surg. Journ.,' 1851, April, 310, 316.

¹⁴ 'Transact. of the Bombay Med. Soc.,' 1837, i, 69.

¹⁵ 'Med. Times and Gaz.,' 1855, Decr., 538.

¹⁶ 'Dublin Journ. of Med. Sc.,' xi, 338.

Phillips. Of 100 children born in India and under ten years of age, who were examined by Jackson, the most indubitable signs of scrofula could be detected in the larger number; among 715 children examined by Spry there were 75 of mixed blood who were all scrofulous, 136 of English parentage who were all healthy, and 504 Hindu children of whom 300 were scrofulous.

In the absence of fuller information from *Further India*, I am unable to say how far Breton's¹ statement that scrofula is rare among the Anamese, may apply generally. But for the *Malay Archipelago* Heymann² says: "Of all the dyscrasias the scrofulous comes to the front most. It is a malady of childhood particularly, so much so that the well-known torpid habit of scrofula may be seen in the Javanese youth wherever we go;" and this account is entirely confirmed in the writings of v. Leent.³ As to the enormous frequency of scrofula in *China* (Tientsin, Fukiang, Chee-foo, Canton, Shanghai, Pekin) there is complete unanimity among observers;⁴ and the authorities for *Japan*⁵ give the same account of it there.

On the *continent of Australia* and in the *islands of the Pacific* scrofula would appear not to have been general until recent times,⁶ or since the natives came in contact with Europeans and in consequence underwent a radical change in their manner of life. In some localities the malady is prevalent to a very great extent, and it has exerted a most injurious influence on the working power of the native population. The regions most affected are the *Hawaiian Islands*,⁷

¹ 'Quelques considér. sur la guérison des plaies chirurgicales . . . chez les Annamites,' Par., 1876.

² 'Darstellung der Krankheiten in den Tropenländern,' 177.

³ 'Arch. de méd. nav.,' 1867, Octbr., 246, 1870, Janv., 14, 1877, Févr., 100.

⁴ Rose, 'Pacific Med. Journ.,' 1862, Octbr.; Wilson, 'Med. Notes on China,' Lond., 1846, 19; Friedel, 'Beiträge,' 62, 69, 126; Morache, 'Annal. d'hyg.,' 1870, Janv., 54; Henderson, 'Edinb. Med. Journ.,' 1876, Novbr., 405; Dudgeon, 'Glasgow Med. Journ.,' 1877, July, 330; Wernich, 'Geogr.-med. Studien,' Berl., 1878, 293.

⁵ Friedell, l. c., 32; Pompe van Meerdervort; Wernich, l. c., 161.

⁶ Scott ('Transact. of the Prov. Med. Assoc.,' 1835, iii, App. xii) met with only occasional cases of scrofula in Hobart Town from 1821 to 1831. Down to a later date Tasmania continued to enjoy a comparative immunity from the malady (Hall, 'Transact. of the Epidemiol. Soc.,' 1865, ii, 85).

⁷ Chapin, 'Amer. Journ. of Med. Sc.,' 1837, May; Gulick, 'New York Journ. of Med.,' 1855, March; Ref. in 'Arch. nav. de méd.,' 1864, Debr., 486.

*Tahiti*¹ and *New Zealand*,² in which last scrofula has wrought frightful devastation among the Maoris. The disease is also met with very frequently in the *Navigator's Islands*,³ the *Tonga*⁴ and *Fiji*⁵ groups, the *Gambier Islands*,⁶ *New Caledonia*⁷ and the *New Hebrides*.⁸

The existence of scrofula on *African soil* is attested by definite information for only a few localities. *McRitchie*⁹ has seen it often among the inhabitants of *St. Helena*; and *Guil*¹⁰ has observed it among the Malagasys in *Nossi-Bé* (north of Madagascar). According to *Livingstone's* statement (which is to be taken with reserve), scrofula is unknown in those parts of Central Africa that lie between the 15th. and 25th parallels of southern latitude. Among the *Hottentots* and *Kaffirs* of *Cape Colony* it is widely prevalent,¹¹ and it is of common occurrence also among the Dutch colonists.¹² Other principal seats of it are in *Abyssinia*, particularly the Red Sea coast around Massowah and the less elevated plains;¹³ and in *Egypt*,¹⁴ where the Georgian and Circassian slaves, the children of the fellahs, and the Turkish children living in the harems, are the greatest sufferers. Also in the adjoining

¹ Wilson, 'Edinb. Med. and Surg. Journ.,' 1806, July, 285; Ref. in 'Arch. de méd. nav.,' 1865, Octbr., 290.

² Swainson, 'On the Climate of New Zealand,' Lond., 1840, 58; Dieffenbach, 'Travels in New Zealand,' Lond., 1843, i, 14, ii, 21; Thomson, 'Brit. and For. Med.-Chir. Rev.,' 1855, April; Tuke, 'Edinb. Med. Journ.,' 1863, Octbr., 221.

³ Turner, 'Nineteen Years in Polynesia,' Lond., 1861; Ref. in 'Arch. de méd. nav.,' 1866, Janv., 32.

Ref., ib., 28.

Ib., 32.

Le Borgne, 'Géogr. méd. de l'archipel des îles Gambier,' Par., 1872.

⁷ Vinson, 'Topogr. méd. de la Nouvelle-Calédonie,' &c., Par., 1858; de Rochas, 'Topogr. méd. de la N.-C.,' Par., 1860, 31; Charlopin, 'Notes rec. en Calédonie,' Montp., 1868, 21; Boyet, 'Arch. de méd. nav.,' 1878, Sptbr., 228.

⁸ Boyet, l. c.

⁹ 'Transact. of the Calcutta Med. Soc.,' 1836, viii, App. xxix.

¹⁰ 'Arch. de méd. nav.,' 1882, Novbr., 330.

¹¹ Black, 'Edinb. Med. and Surg. Journ.,' 1853, Apr., 256; Scherzer, 'Zeitschr. der Wiener Aerzte,' 1858, 152; Schwarz, ib., 659.

¹² Schwarz, ib., 630; Kretschmar, 'Südafrikanische Skizzen,' Leipz., 1853.

¹³ Bruce, 'Travels,' Ger. ed., iii, 32; Petit, in Lefebure's 'Voyage,' Pruner, 'Krankheiten des Orients,' 321; Courbon, 'Observ. topogr. et méd.,' &c., Par., 1861, 37; Blanc, 'Gaz. hebdom. de méd.,' 1874, 349, Feuill.

¹⁴ Pruner, l. c.; Ref. in 'Arch. de méd. nav.,' 1869, Mai, 326.

negro countries, as well as in *Tunis*¹ and *Algiers*,² scrofula takes one of the first places among the chronic disorders of nutrition. I am not acquainted with any trustworthy information as to its occurrence in *Senegambia*;³ but it is said by Ballay⁴ to be a very general malady in the Ogowai country (region of the Gaboon, on the western side of the continent one degree south of the equator); and it would appear from Clarke's⁵ way of speaking to be anything but rare on the *Gold Coast*.

The information before us from the *Western Hemisphere* is also insufficient to furnish a tolerably complete outline of the distribution of scrofula there. From the most northern latitudes we have Blaschke's⁶ account of its great frequency among the native children in *New Archangel* (Alaska); and Gras⁷ tells us that in Miquelon (*Newfoundland*), the home of cod-liver oil, scrofula contributes not a little to the mortality among children. In *Greenland*, on the other hand, we have it on the authority not only of Lange's⁸ own experience but also of the observations of several of his predecessors in office, that scrofula is extremely rare or even unknown. In the *United States* it must now be as prevalent as in Europe. There is a noteworthy remark of Hildreth's,⁹ dating from the year 1830, to the effect that the diffusion of the malady had kept pace with advancing civilisation, proceeding from east to west;

¹ Ferrini, 'Saggio sul clima . . . di Tunisi,' &c., Milano, 1860, 216; Rebatal and Tirand, 'Lyon médical,' 1874, Nr. 13, 249.

² Cambay, 'Mém. de méd. milit.,' 1842, lvii, 1; Bertherand, 'Med. et hyg. des Arabes,' Par., 1855; Arnand, 'Méd. et hyg. des pays chauds,' &c., 417; Challan, 'Gaz. méd. de l'Algérie,' 1868, 116; Creissel, 'Mém. de méd. milit.,' 1873, 369; Claudot, ib., 1877, 194. The statements of Bertrand (ib., 1867, Mars., 199) and Bazille ('Gaz. méd. de l'Algérie,' 1868, 30) as to the rarity of scrofula among the Arab children, particularly as regards Kabylia, have been entirely overthrown by the observers above quoted (see the chapter, in the next volume, on "Maladie du sommeil").

³ Chassaniol ('Arch. de méd. nav.,' 1865, Mai, 507) says that he did not see it often; but Corre (ib., 1877, Mai, 330) says that scrofula is exceedingly common among the natives on the coast of Senegambia.

⁴ "L'Ogooué" ('Afrique équatoriale occidentale'), Par., 1880, 39.

⁵ 'Transact. of the Epidemiol. Soc.,' 1860, i, 104.

⁶ 'Topogr. med. portus Novi-Archangelensis,' Petrop., 1842, 62.

⁷ 'Quelques mots sur Miquelon,' Montpell., 1867.

⁸ 'Bemaerkninger om Grönlands Sygdomsforhold,' Kjöbenhavn, 1864, 27.

⁹ 'Amer. Journ. of Med. Sc.,' 1830, Febr., 329.

"scrofulous affections are more frequent," he says, referring to Ohio, "than they were formerly; and will probably continue to increase as the country becomes more highly cultivated and people more luxurious in their habits." Along with this we may take the fact which is vouched for equally by Moses¹ and by Glisan,² that scrofula did not exist among the Indians of Oregon Territory until they began to be more and more confined in their hunting grounds, to settle at particular spots, to adopt the customs and vices of the white man, and to degenerate under the influence of an entirely altered manner of living. In California also, where Praslow³ met with little of it at the time of the first outflush of prosperity in the State, it is now very prevalent (according to Lantoin⁴), so far at least as relates to San Francisco. At Monterey, which is a long way from the gold fields and escaped the rush of adventurers, the disease was but little prevalent even in 1853; but there is no later intelligence of it from that part of California.

In Vera Cruz, on the *Mexican* coast, Heinemann⁵ has often seen scrofula among the children of mixed parentage and of the white race; but on the table-land of Mexico (Anahuac) it is rare.⁶ From *Central America* I know of only one notice⁷ of scrofula, to the effect that it is often observed in *Guatemala*. For the *West Indies* we have the older accounts of scrofula in Jamaica by Armstrong⁸ and Lemprière,⁹ the former testifying to its common occurrence among negro children, and the latter stating that it was much rarer than in England and of a very mild type. Among recent authorities, Ruz¹⁰ says for Martinique what Lemprière had said for Jamaica; whereas Goës¹¹ for St. Bartholomew, Hamon-

¹ *Ib.*, 1855, Jan., 32.

² *Ib.*, 1865, Jan., 79.

³ *L. c.*, p. 56.

⁴ 'Arch. de med. nav.,' 1872, Mars.

⁵ In 'Virchow's Archiv,' 1873, lviii, 178.

⁶ Jourdanet, 'La Mexique,' &c., Par., 1864, 4:2; Coindet, 'Mém. de méd. milit.,' 1869, Avril, 273.

⁷ Durant, 'Arch. de la méd. belge,' 1846, Mai.

⁸ In Duncan, 'Annals of Med.,' 1802, vi, 370.

⁹ 'Observations on the Diseases . . . in Jamaica,' Lond., 1799, i, 45.

¹⁰ 'Arch. de méd. nav.,' 1869, Novbr., 349.

¹¹ 'Hygiea,' 1868, Octbr., 460.

Dufougeray¹ for St. Martin, and Jackson² for Barbadoes are of one opinion that the malady is prevalent generally throughout these islands. It is not to be denied that these discrepancies in the statements of authorities in the West Indies may perhaps depend on the scantiness of the observations; but it is possible, also, that they may be due to real differences in the several localities. It may be the same with the discrepant accounts of the prevalence of scrofula in *Brazil*. Sigaud's³ opinion is that "les scrophules sont remarquables par leur rareté;" while Rendu⁴ assures us that "les scrofulos . . . sont des affections très fréquentes au Brésil;" and Tschudi⁵ speaks of its endemic prevalence in the northern provinces, while Plagge⁶ asserts the same for the province of Maranhão, and Rey⁷ mentions the frequent occurrence of the disease in Santa Catharina, particularly in the up-country districts. In the *River Plate's States* (Argentine Republic), particularly in the large towns (Monte Video, Buenos Ayres), scrofula is rarely seen among the whites but more commonly among negroes and mestizzos, according to Mantegazza⁸ and Rey, who are corroborated by Saurel. In *Paraguay* also it is rare.⁹ But in *Chili*¹⁰ and *Peru*¹¹ it has attained to a very considerable diffusion, and, as Tschudi tells us, not only along the coast, but also in the highest mountain towns; even in Cerro de Pasco, at a height of 13,500 feet, he found many scrofulous persons. In *Ecuador* (Guayaquil) scrofula holds a prominent place among chronic diseases.¹²

¹ 'Arch. de méd. nav.,' 1883, Jan., 57.

² 'Boston Med. and Surg. Journ.,' 1867, July, 448.

³ 'Du climat et des maladies du Brésil,' Par., 1844, 424.

⁴ 'Etudes topogr. et méd. sur les Brésil,' Par., 1848, 81.

⁵ 'Oest. med. Wochenschr.,' 1846, 472.

⁶ 'Monatsbl. für med. Statist.,' 1857, Nr. 10.

⁷ 'Arch. de méd. nav.,' 1877, Janv., 27.

⁸ 'Lettere mediche sulla America meridionale,' Milano, 1860, i, 14, 19.

⁹ Mantegazza, ib., i, 285.

¹⁰ Brandin, 'De la influencia de los diferentes climas del universo sobre el hombre,' &c., Lima, 1826; Gillis, in 'Deutsche Klinik,' 1856, Nr. 24; Fischer, 'Arch. de méd. nav.,' 1864, Juill, 21; Duplouty, ib., Août, 108; Ullersperger, in 'Virchow's Archiv,' 1869, xlviii, 501.

¹¹ Tschudi, l. c.; Duplouty, l. c., Sptbr., 189.

¹² Duplouty, ib., Octbr., 282.

§ 214. INFLUENCE OF CLIMATE AND CHANGE OF RESIDENCE.

Although our knowledge of the geographical distribution of scrofula remains incomplete, and although for the present we are not in a position to find a numerical expression for the frequency of the disease in the various parts of the world,¹ which would give us the only safe means of making a comparison ; yet there can be no doubt at all that the malady is, as we said at the outset, a decidedly ubiquitous one, and that the *conditions of climate* proper to the various latitudes of the globe exert no decisive influence either on its existence or on its amount. The prominent place that scrofula takes among the national maladies of India, the East Indies, the southern coasts of China, and the tropical provinces of Brazil and Peru affords evidence that the disease spares the equatorial regions just as little as the temperate and high latitudes ; while its comparatively rare occurrence in the States of the River Plate, in Greenland, in the Farøe Islands and elsewhere, teaches us that its prevalence stands in no necessary association with a temperate or cold climate. The slight influence of the climate of a locality upon the amount of scrofula is further shown by the very unequal distribution of the disease at the several points within large districts which present no material differences in regard to their climate ; as well as by the fact that at other points, where there are such climatic differences, as on the central plateau of Spain compared with the Mediterranean coast, scrofula is uniformly common ; and lastly by the observations above mentioned, that in some considerable territories the malady did not appear, or did not spread much until modern or quite recent times, such territories having previously enjoyed a marked exemption from it.

There is one respect in which the influence of climate does

¹ The tables of mortality do not afford materials suited to that purpose. Not to mention the unavoidable errors of diagnosis, doubtless even of a gross kind, which run through them, there is, in regard to scrofula in particular, the fact that in many of these lists rickets is mixed up with it, while cases ending fatally in scrofulous affections of the bones are placed under diseases of the osseous system.

appear to have an important significance for the development of the disease. It is shown in the fact that an unusually large number of persons who had come from lower latitudes, the tropics especially, into colder regions, are attacked by scrofula, all the more speedily and the more severely the greater the difference between the climate of their new and their old home.

"I have observed," says Lugol,¹ "that the residents from countries within the tropics exhibit the saddest traces of the effect of our temperate climate upon them; scrofulous disease develops in them with unusual rapidity." Prichard² met with many facts confirming that observation among persons born in Brazil or the Southern States of the Union, who came to Paris.

Cooper, writing of England, says:³ "People from the East or West Indies, who come over to this country, not unfrequently fall a prey to scrofulous disease. Many children born in the East and West Indies are sent to this country to be educated, and therefore we have an opportunity of seeing the effect of climate on their constitutions; and I can assure you, that it frequently requires the greatest possible care to save them from the danger of scrofulous disease of the joints and absorbent glands, and very often, with all your care and attention, they will die of scrofulous disease. Those of the West Indies less frequently die of scrofula than persons from the East Indies; but I have seen some from the South-Sea Islands, and most of them have died from scrofulous complaints."

Pearson,⁴ who had medical charge of an institution for persons brought from the West Coast of Africa to England, says: "It is remarkable that boys brought from tropical climates, from the age of eight to twelve, almost uniformly become scrofulous. They bear the first winter tolerably well, but droop during the second, and the third generally proves fatal to them."

It cannot well be denied, as a general truth, that weather influences which are absolutely bad, or (as in these cases) relatively so, and which make themselves felt most at the age of childhood, may lower the body's power of resisting noxious influences in general, and so create a predisposition for the development of scrofula. But in considering the frequency of attacks among persons arrived from foreign parts, we have to keep in mind also, that in changing their climate

¹ L. c., p. 231.

² Art. "Scrophule," in the 'Dictionnaire de Médecine.'

³ 'Lancet,' 1824, iv, 65.

⁴ 'Annual Med. Rev.,' ii, 130.

they at the same time make material changes in their mode of living; and these changes must not only weigh in the scale in our estimate of the pathogenesis, but they are doubtless a factor of especial importance therein.

§ 215. QUESTION OF THE INFLUENCE OF A WET SOIL.

In support of the idea that *the altitude* has a direct influence on the occurrence and diffusion of scrofula, there is not a single piece of evidence from any side. The rarity of the disease on the Anahuac (table-land of Mexico), and at some elevated points in the Swiss and Italian Alps and in the Vosges, is accounted for not by their altitude itself, but by other causes; for at many other equally elevated and even more elevated points scrofula is met with in as general diffusion as on the plains and in not less numerous cases.

We find examples of this in the prevalence of the malady at some places in the Saxon and Bohemian Metal-mountains, in Upper Austria, and in the Alps and the Jura of Canton Vaud, where, as Lebert tells us,¹ the number of the scrofulous never failed to strike him in his numerous excursions to the mountains. Other instances are the prevalence of the disease on the elevated table-land of Mysore, in the highest passes of the Himalaya in Kumaon (Mc Clelland), and in the Peruvian Andes at elevations of 3000 to 4000 metres (10,000 to 13,000 feet).

There is just as faint a connexion to be discovered between the existence or amount of scrofula and particular *geological conditions*. Escherich,² who reasons on the basis of a few facts and whose premises (*e. g.* the genetic connexion of scrofula with goitre and cretinism) are erroneous, has adopted the opinion that the disease occurs as one proper to the locality, and not as an acquired habit, on the older rocks only (including the primary, the transition, and the mesozoic formations up to the chalk), while it exists in mere sporadic cases on the tertiary and recent deposits. This idea is overthrown by the wide diffusion of scrofula on the alluvial and diluvial soils of Holland, Belgium, North Germany and other countries. A geological map which I have used to elucidate

¹ L. c., p. 48.

² 'Allgem. Zeitschr. für Chirurgie und Heilkde.,' 1843, No. 30.

the question at issue, serves to show that more or less considerable centres of scrofula can be made out on almost every geological formation, and that no formation has a preference either way over any other.

The views of observers diverge as to the influence of a *wet or swampy soil* on the production of scrofula; and it is this very difference of opinion, with the arguments adduced on either side, that provide us, to my thinking, with the correct means of estimating the etiological importance of the factor in question. The accounts of the general diffusion of scrofula in some districts of Sweden,¹ in the damp low-grounds of Oldenburg² and Holland,³ in the wet or swampy valleys of Upper Austria⁴ and Styria, and in the plain of Lombardy,⁵ lay quite special stress upon that factor in the pathogenesis; whereas other observers tell us that the disease within the limits of their own practice is actually more common on dry soil than in damp localities in the vicinity.⁶ Some Belgian authorities, such as Waldeck, would assign the draining of the ground as one of the determining causes of the occurrence of scrofula, and would discover a causal connexion between such changes in the soil and the notable increase of the malady which was observed in many parts of Belgium about the period from 1830 to 1840. The fallacy of this conclusion has been aptly exposed by Meynne,⁷ in adducing proof that the increasing misery of living among the poor of the country in that very period was the real cause of the phenomenon; “depuis cette époque les salaires ont diminué, une grande misère est survenue parmi les tisserands, fort nombreux dans ce canton (Ecloo); l'alimentation s'en est ressentie, elle est

¹ Huss, l. c., p. 69.

² Goldschmidt, in ‘Häser’s Arch. für die ges. Med.,’ 1845, vii, 308.

³ See Büchner, ‘Bijdrag tot de geneesk. Topogr. van Gouda,’ Gouda, 1842.

⁴ Streinz, ‘Oest. med. Jahrb.,’ 1831, Neueste Folge, i, 4, Heft 46.

⁵ *Ib.*, l. c.

⁶ In some wet and (here or there) swampy districts of the western and southern coasts of France, and in some swampy localities in the department of the Somme, scrofula is rarer than in Rheims and Orleans, which are built upon a dry bottom, or in Montpellier, which is situated on hilly ground. Beaconsfield (Bucks), according to Rumsey (‘Transact. of the Prov. Med. and Surg. Assoc.,’ 1844, June), although it lies low and is damp, has not nearly so much scrofula as some adjoining districts where the soil is dry.

⁷ L. c., p. 157.

devenue insuffisante pour beaucoup d'ouvriers, suffisante mais grossière et exclusivement végétale pour la généralité." Lugol, guided by ample observations made in France, pronounces very decidedly, and it appears to me with perfect justice, against the doctrine that the amount of scrofula stands in any definite relation to the wetness or dryness of the soil.

"Brittany," he says,¹ "is a damp region, but scrofula is not endemic in it; and if it does present itself in that character at a few places within the province, these are not the dampest localities. In no locality is the disease so common and of so severe a type as in the dry country of Champagne. . . . In the Pyrenees, we find particularly striking contrasts in the nature of localities where scrofula is endemic. Let us take as an example a village on the banks of the Adour. The water in that stream runs at a level with the huts built on the bank; considerable quantities of it flow around the dwellings and into the gardens, in which the prevailing colour is the green of a landscape. The dwellers in this spot are scrofulous . . . a fact which may seem at the first glance to confirm the notion that dampness is the cause of endemic scrofula, all the more so that the huts which stand a little way back from the bank and are a few metres above the water-level are occupied by a far finer set of people in every respect. The higher we go, the better the type of inhabitants. But at a point still higher above the stream, in a dry and pure region, we come suddenly upon an unexpected thing—the residents at the top of the mountain are scrofulous. Thus we find scrofula to be endemic at places which are entirely different from those on the river or situated at corresponding elevations. These contrasts, which may be often seen in the Pyrenees within a short radius as well as in other countries where scrofula is endemic, are against the idea that dampness or any other condition of locality is the cause that produces an endemic of scrofula."

However, in discussing this question, we ought not to overlook the consideration that dampness of the ground has a definite effect on the social condition of the people as well as on the conditions of climate, and may accordingly exert in the last resort an influence, if only a remote one, on the occurrence of scrofula.

¹ L. c., p. 216.

§ 216. EVIDENCE THAT IT IS DUE TO DIETETIC AND HYGIENIC ERRORS.

In the opinion of nearly all observers, the real cause of the scrofulous disease is to be looked for in some error of nutrition and manner of living ; although as yet no agreement has been arrived at among them whether it is a matter of some noxious influence of a definite kind, or whether various injurious things, rooted in a defective hygiene and acting singly or in combination, may not furnish the pathogenetic factor. Another open question is how far the attack of illness depends upon individual predisposition or congenital morbid diathesis.

The social positions of *various classes of the population* as fixed by a certain measure of comfort or by the possession of the bare necessities of life, do not reveal on the large scale any real differences in the amount of scrofula. Even if the disease is, under certain conditions to be given in the sequel, rather more common in towns than among the country population, yet it is no stranger to the latter ; and whoever has had occasion, like the writer of these lines, to study the state of health among the inhabitants of country districts, cannot but confirm the opinion of Lebert, Phillips and others, that scrofula takes, under those conditions as well as under their opposite, one of the foremost places among the prevalent maladies of childhood and youth. Unfortunately the statistics do not afford the means of proving this mathematically, for reasons that have been several times stated. Still less do they help us to estimate the number of cases among *the well-to-do and the indigent* respectively. But in that matter also the daily experience of practitioners discovers no important differences ; and it is only when there is a concentration of the influences favorable to the origin of the disease pressing uniformly upon one considerable section of the population that the predominance of scrofula is seen to be dependent on circumstances of living, as will appear in the sequel.

The cause of the disease must be sought, accordingly, in such influences as are universal in their nature, are generally

diffused over the globe, and make themselves felt equally among all classes. Among these a principal factor, according to experience, is the more or less *unsatisfactory way of feeding infants and very young children*. This doctrine, which has been firmly held by the practitioners of all times, finds very decided expression in all the accounts before me from the most various parts of the world. From no quarter has it met with opposition; neither is it weakened by the consideration that scrofula, as we have seen, is a disease not of the proletariat only, but just as much of the children of the better situated and well-to-do classes.

“The frequency of scrofula amongst the classes of society who live in wealth or comfort,” says Phillips¹, “has been supposed to militate against any view of the disease which assigned to insufficient food or improper feeding a large share in the production of the disease. But diseased nutrition may co-exist with sufficiency of food and even with seemingly judicious feeding, and is, perhaps, as frequently found in the pampered child of luxury as in the cottage of the peasant.”

A second factor in the pathogenesis, which many observers would rate as of specially high importance, is *the want of exercise for children in the open air, or the constant keeping of them indoors, especially where the ventilation is bad and the atmosphere charged with organic effluvia*. In some quarters this noxious influence is even made out to be the true cause of the disease. Thus Alison,² from his experience of the Edinburgh Dispensary, says: “I am thoroughly convinced, from the amount of it (scrofula) that I have seen in families not suffering under any material privations, that it depends much more on want of pure air and exercise, than on deficient nourishment.”

To the same effect are the opinions of Cooper,³ Eager,⁴ Byford⁵ and others; while Baudelocque,⁶ from his experience

¹ L. c., p. 242.

² ‘Transact. of the Edin. Med.-Chir. Soc.,’ 1824, i. 397.

³ L. c., 72.

⁴ ‘Dublin Journ. of Med. Sc.,’ 1834, July, 347: “I have abundant reasons to think that the absence of direct solar rays and a long sojourn in a confined atmosphere contribute more than any other towards the development of scrofula.”

⁵ ‘Transact of the Amer. Med. Assoc.,’ 1855, viii.

⁶ ‘Revue méd.,’ 1832, Jan., p. 10; and ‘Études sur les causes . . . de la maladie scrophuleuse,’ Par., 1834.

of the Hôpital des Enfants in Paris, lays particular stress on this factor as “cause principale de la maladie scrofuleuse, une cause qui domine toutes les autres, et sans laquelle peut-être la maladie ne se développerait jamais, ou au moins serait très-rare.”

Even Lugol,¹ who refers the spread of scrofula exclusively to heredity, assigns to the injurious influence in question a prominent place among the contributory causes.

But against the somewhat exclusive pre-eminence that Baudelocque has given to this etiological factor, we have to place the circumstance that scrofula is far from rare, as we have seen, among the population of many country districts, notwithstanding that the children pass the time in running about in the open air. In many such localities, indeed, it is very widely spread, as in the mountainous parts of the Canton Vaud, according to Lebert's observations. On the other hand there is a large number of facts which put it beyond doubt that want of fresh air and exercise has an importance for the development of the malady that is not to be underrated. We have first of all the predominance of scrofula among the children of the poor and of the working class in the great centres of industry and trade, especially in the large manufacturing towns; on which point there is but one opinion among the authorities.²

Next there is its prevalence in the mountain villages of Bohemia, Saxony and the Harz, inhabited by spinners, weavers and stocking-workers, who are put to these occupations at a very early age and who ply their industry in small and dull rooms.³ Again we find the malady extremely common in

¹ ‘Gaz. des hôpit.,’ No. 71; and ‘Recherches, &c.,’ p. 240 (Germ. ed.).

² See the ‘Sanitätsbericht des Medicinal-Collegiums von Westfalen,’ 1845, p. 45, with reference to the diffusion of the disease in the manufacturing towns of Westphalia; and the account given by Meyne (l. c., p. 487), for Belgium.

³ See Cartellieri's statements (‘Oest. med. Jahrb.,’ 1843, ii, 354) with reference to the Circle of Leitmeritz, in Bohemia; and Klinge's for Andreasberg, in the Harz, where even the little girls are employed in making bone-lace. Black (‘Transact. of the Prov. Med. and Surg. Assoc.,’ 1832, v, 179) remarks of the state of health among the young persons employed in the spinning-mills of Bolton: “They are more peculiarly liable than other classes of the operatives to the different kinds of scrofula affecting girls and boys in the joints and glands.” The same view is taken by Hinder (‘Med. Times and Gaz.,’ 1854, Feb., p. 54) of the causes of the enormous amount of scrofula among the children of the shawl-weaving popu-

the coal districts of England, Scotland,¹ and the North of France. Under all these circumstances there is an *ensemble* of harmful influences to be allowed for; and it remains a question, accordingly, how much importance is to be assigned to any one of them. But apart from such cases, the importance of the particular factor that now concerns us can be shown in a very convincing manner in cases where the disease either breaks out generally among all groups of the population, other noxious influences, such as of diet, being excluded; or, where the conditions are in other respects the same, but the malady falls mostly on that one section which is principally or exclusively subject to the injurious influence in question. Of that sort is the evidence furnished by the state of health among children or young persons in foundling hospitals and orphanages, in workhouses, homes of industry and such like institutions. Out of a large number of observations of that kind I give here a few of the most interesting and most convincing.

In the Educational Institute of St. Petersburg, according to Doepp, there were, from 1830 to 1833, among pupils of ten to twenty-three years of age, four times as many cases of scrofula in girls as in youths. The reason of this could not be either the food, which was the same for all, or the lodging, which was even better for the girls than for the boys and young men; it could only be that the female pupils, yielding to their incorrigible propensity for a sedentary life, would stay constantly in their small rooms in the Institute, and could not be induced, whether by exhortation or by discipline, to resort to the large recreation-rooms or to spend part of their time in the garden of the institution. Blatin states that, in the Training College of Billodes near Locle (Neuchâtel), where the pupils are kept shut up in rooms almost constantly, they are, without exception, scrofulous. Glover² adduces the fact published by Tyler Smith,³ that in a workhouse in Kent the whole of the seventy-eight boys and all but three of the ninety-four girls, were found to be suffering from scrofula, although only a few of them had shown signs

lation of Umritsur, in Cashmere. He points out that while the adults are employed in sorting and spinning the wool, children of eight years and upwards are busy the whole day over the loom in buildings that are overcrowded although open, and that when work is over they spend their time in dull, filthy and unventilated rooms.

¹ Alison, 'Lancet,' 1842, i, 800.

² 'On the Pathology and Treatment of Scrofula,' Lond., 1846, Germ. Transl., Berlin, 1847, p. 134.

³ 'Scrofula, its nature, &c.,' Lond., 1844.

of the disease before they were admitted into the institution. Glover inquired also into the number of cases of scrofula in three workhouses, among whose inmates there were altogether 164 persons below the age of fifteen: the first, in a large manufacturing town, had 53 children with scrofula out of 112, or 47 per cent.; the second, in a seaport, had 9 cases of scrofula out of 35, or 26 per cent., this being the proportion for those who lived continuously in the house, whereas among 20 who only came to school there was not a single case; in the third, a country workhouse, there were 7 children out of 18, or 39 per cent., who were more or less scrofulous. Fourcault¹ mentions the following fact with reference to children in the workhouse attached to the General Hospital of Lille, of whom a large number of both sexes, foundlings and orphans, had been brought up previously in the country, and were taken in as soon as they were able to work: the girls occupy large and well-ventilated rooms, where they busy themselves with female handiwork; the boys take service with artificers in the town. The latter keep in good health, but large numbers of the girls develop a general feebleness of habit, chlorosis, and the like, and many of them die of scrofula. Hall observes that scrofula is, generally speaking, rare in Tasmania, but that it is seen very often in the children who are inmates of the overcrowded and badly-maintained orphanages in the colony. "In the Department of Lozère," says Alibert,² "a third part of the population is occupied in the manufacture of a woollen stuff known as 'serge de Mende.' That is the one industry of the country, and the chief source of livelihood of the inhabitants. The wool is worked without oil; and to facilitate the working of it, the industry is carried on in low, vaulted rooms, very damp and hot. It is this class of work-people that supplies the largest contingent of scrofulous persons in the Department." Bredow³ gives the following observations on the development of scrofula among the juvenile hands (eleven to twenty-two years old) in the woollen-spinning division of the Imperial Alexandrowski Factory, and in a private silk-mill. In the Imperial Factory there were 666 hands between the ages mentioned; of these 360 lived at the works, and 32 of them, or 9 per cent., acquired scrofula after entering; 217 lived outside the factory, and 29 of them, or 13 per cent., became scrofulous subsequent to joining; and of the remaining 89, who lived in villages several versts distant from the factory, only 2 had the disease, or less than 2 per cent. Of 162 persons of the same ages in the private silk factory, 63, or 40 per cent., became scrofulous during the time they were employed in it. We have to bear in mind here that in both factories there was nothing wanting in cleanliness, good clothing, and proper food; but that (1) the young hands in the Imperial Factory spent the leisure time allowed them in running about and playing in the open air, while the boys in

¹ 'Causes générales des maladies chroniques,' Paris, 1844.

² 'Precis théorique et pratique sur les maladies de la peau,' Paris, 1818, ii., 364.

³ 'Preuss. med. Vereins-Ztg.,' 1845, No. 45.

the private factory spent most of the corresponding intervals in their dull sleeping places or in lolling lazily upon their beds; (2) those of the hands who had to go several versts every day to and from the factory enjoyed the best health; and (3) the worst cases were those who, after being used to the open air, hardly ever left the gloomy purlieus of the factory, except to go to their dormitories.

Lastly, I shall refer to Dr. Baly's report (as given by Phillips)¹ on the occurrence of scrofula among the prisoners confined in Millbank Penitentiary; although, as Baly himself regrets, it affords no perfectly clear view of the circumstances in question, for the reason that scrofula, in the table of sickness, is entered under the same heading with pulmonary consumption, and it is possible only within limits to separate out the cases belonging to the latter disease. According to the figures there were, in 1840, 14 per 1000 attacked with scrofula among 1052 prisoners, and, in 1844, 13·5 per 1000 among 3249 prisoners. (It has to be observed that for those who were received into the prison with the first signs of scrofula upon them, the origin of the malady might be referred to former imprisonments.) Among the prisoners received in 1840 were 510 female convicts under sentence of transportation who remained on an average three months at Millbank, and only two cases of scrofula developed among these; whereas among 520 others admitted the same year, who remained in the prison from two to three years, no fewer than 78 were affected with scrofula and pulmonary consumption before the end of their term of imprisonment. It is noteworthy that the number of cases rises in proportion to the duration of the imprisonment; so that in 1000 prisoners, 6·9 either died or were released on account of the disease in their first year, 31·32 in their second, 49·9 in their third, 52·38 in their fourth, and 63·83 in their fifth. Baly expressly says that there was no reason to look for the cause of these attacks in the kind of diet, inasmuch as the diet of Millbank Penitentiary, since the unfortunate experience of 1824 (see p. 550), left nothing to desire; and that the cause was more probably to be assigned exclusively to the want of exercise in the open air, or, in other words, to living in an atmosphere rendered impure by organic effluvia.

An impartial survey of all the facts here given, which are corroborated by the observations above referred to on the causes of the outbreak and spread of scrofula among the natives of New Zealand, Oregon and other countries, should lead us, in my view, to the conviction that both etiological factors (diet and close confinement) play a prominent part in the pathogenesis. It is all the more difficult to say which of them is the more important, for the reason that they often act side by side as causes of disease.

¹ L. c., p. 362.

The history and geographical distribution of scrofula teach us, further, that no race or nationality enjoys an absolute or even relative immunity from it, that the malady becomes prevalent among all communities and families whenever the etiological factors favorable to its development make themselves felt. Some observers, such as Marpurgo, Vauvray and others, assure us that the disease is rare among the nomade Arabs ; but the explanation of that is not their nationality, inasmuch as their own stock are by no means exempt from scrofula in towns and settlements in Algiers. Again, if the malady is not often seen among the nomade Kirghiz,¹ the reason of that is that weakly children are regarded by them as a punishment from heaven, and perish from want of tending before scrofula has time to develop.

§ 217. TO A GREAT EXTENT INHERITED, BUT NOT NECESSARILY FROM SCROFULA IN THE PARENTS.

One of the most intricate points in the etiology of scrofula is the question of the hereditary transmission of the disease. It is altogether denied or very much doubted by some (Phillips, Rilliet and Barthez, and others), who are supported by few observations, or by very faulty statistics, or by erroneous assumptions ; but by far the larger number of observers at all times have pronounced decidedly for hereditary transmission. Among others Cooper says : “ That scrofula is a hereditary disease appears as clear to me as can be ; and they who deny it, deny the evidence of their senses.” A few, such as Lugol and Guet, have even gone so far as to make the inherited disposition a *conditio sine quâ non* for the development of the malady. “ Pour nous,” says Guet,² “ un enfant naît scrofuleux, mais les circonstances qui viennent se grouper autour de lui après sa naissance peuvent hâter ou retarder ou même quelquefois arrêter le principe qui lui a été

¹ This statement of Maydell for the Kirghiz Steppe is one that I cannot confirm from my own observations made (to a very limited extent however) in the Government of Astrakhan. In the kibitkas of the Kirghiz, loaded with filth, I have seen several children with typical scrofula.

² ‘Revue méd.,’ 1884, April, p. 533.

transmis par l'hérédité: ce n'est donc que d'une manière secondaire que nous comprenons le mode d'action des causes énoncées plus haut."

At the present day there is really no doubt that scrofula may develop quite independently of hereditary conditions, and under the noxious influences above mentioned; but heredity forms an especially prominent factor in its evolution. The only differences of opinion are whether the inherited diathesis of the children is always to be referred to scrofula of the parents, or whether it may not be sometimes the consequence either of other diseases in the progenitors, such as syphilis,¹ or cancerous disease, or of conditions of ill-health due to immaturity or advanced age, or of drunkenness, or of the marriage of near kin. As to the nature of the diathesis, also, there is for the present much obscurity, some discovering it in irregularities of the blood and lymph, from the humoral point of view, while others, from the side of solidism, find it in an abnormal state of the lymphatic glandular system, and still others in the transmission of an organic (parasitic) germ. When the disposition is highly developed, opportunities which are slight in themselves may serve to bring out the disease; and this is how we explain the frequent occurrence of scrofula in well-to-do families, in which there seem to be present all the conditions necessary for the healthy development of the young, but in which the disease passes by inheritance from generation to generation.

§ 218. NO EVIDENCE THAT IT IS INOCULABLE ALONG WITH
VACCINIA OR OTHERWISE.

Against the interpretation of scrofula that we have given up to this point, as a disease, namely, that depends upon

¹ An opinion specially held by Otto for Denmark, Briard for Ancona, Moris for Sardinia, King ('Med. Gaz.,' v, 805) for England, Courbon for Abyssinia, and a number of authorities for the southern United States and for the islands of the Pacific that have had particularly severe visitations of late. There are fallacies or errors of diagnosis underlying the doctrine formerly held by Astruc, Hufeland, Alibert and others, and lately revived by Rabatel for Tunis (although long ago overthrown by Kortum), that congenital syphilis under some circumstances takes the form of scrofula, or that, as Rabatel says, there is "transformation par l'hérédité d'une autre diathèse, de la syphilis."

chronic errors of nutrition, there was a theory developed in the first scientific treatises on the malady to the effect that it owed its origin to a *miasma*, or that it was to be counted among the *infective diseases*, spreading by way of *contagion*. Borden¹ was the first to formulate definitely the doctrine of the miasmatic-contagious character of scrofula: "Il existe donc dans la nature une sorte de *miasme scrofuleux*," he says, "qui est sans doute formé quelquefois par les révolutions qui arrivent aux différentes humeurs, et qui peut fort bien, en passant d'un sujet à l'autre, aller, comme le levain dans la pâte, gâter des humeurs saines; mais il faut qu'il trouve une disposition particulière dans le sujet pour y agir; il a besoin d'y être mis en action par un certain jeu des organes, et par l'état particulier des liqueurs."

Contemporary and later writers, such as Charmetton, Pujol,² Baumes³ and others, held fast to the belief in a contagious transmission of the malady, tacitly accepting the assumption of a "scrofulous virus;" and latterly there has been no lack of hypotheses of transmission by the milk of scrofulous nurses, by the milk and flesh of tuberculous cows, by vaccination, and so forth; nor of speculative attempts to prove the *parasitic* nature of the hypothetical virus, which latter may appear to have found positive support in the discovery by Koch of the so-called bacillus of tubercle. From the clinical point of view, or following the statistics of practical experience, the notion of a specific morbid poison of scrofula, communicable by contact or *per distans*, has no warrant whatsoever. The number of cases where the disease has developed in persons who had, before their illness, been brought into more or less intimate and prolonged contact with scrofulous subjects (assuming that the facts may be taken to justify the conclusion *post hoc ergo propter hoc*), is so insignificant in proportion to the enormous number of the scrofulous who are continually in the closest intercourse with their whole circle, and that sometimes a very large circle—I am thinking of scrofulous children at school—without in the

¹ L. c., 74. In 'Oeuvres complètes,' Par., 1818, i, 442.

² 'Oeuvres diverses de méd. prat.,' Castres, 1802, iii, 1.

³ 'Traité sur le vice scrophuleux, &c.,' Par., 1805.

least imperilling their associates,¹ that no impartial observer² can seriously entertain the idea of concluding for the spread of the disease by contagion. But even these isolated cases, when closely looked into, lose all the value assigned to them as evidence of that doctrine. They are mostly cases of illness among children of the same family, who do not fall ill all together, but one after the other as they reach a certain age ; and they are not by any means all the children of the family, but only some of them, the other brothers and sisters remaining in good health. Foundling hospitals, orphanages, training institutions and the like, where there is ample means of observing how the disease spreads, do not furnish a single report in which it is even hinted at that the disease had been propagated among the juvenile inmates by way of contagion. On the other hand some of the authorities, such as Baudelocque for the Hôpital des Enfants, and Pinel and Richerand for the Hôpital St. Louis, explicitly deny this mode of diffusion. Baumes says that scrofulous nurses may give the disease to their sucklings, but he is in our debt for a proof that they have ever done so. The assertion that the milk of tuberculous cows induces scrofula rests upon mere hypothesis ;³ not a single well-authenticated case of that kind has occurred, whereas it can be shown that the whole population of villages have eaten the meat of tuberculous cattle for years together (and doubtless drunk the milk of tuberculous cows) without any increase in the amount of scrofula having been remarked among them.⁴

It cannot be denied that children fall into scrofula from time to time after vaccination ; but there is no evidence

¹ "Quotidie occurrunt exempla," says Kortum (l. c., 216), "ubi sani infantes cum scrofulosis arcto et ipsius lecti consortio fruuntur, nec tamen ipsis morbus communicatur."

² I do not express my own conviction merely, but the conclusion deduced from the experience of many busy and observant practitioners with whom I have conversed on the question.

³ It is well known that the pearl-disease of cattle has been identified with tubercle (and scrofula) of the human subject [but only in the ultimate microscopic analysis].

⁴ See the interesting paper by Schottelius, in 'Virchow's Archiv,' 1883, xci, 136. The fact stated in the text I have on the authority of a thoroughly trustworthy medical officer ; and other observations of the same kind have been made on an extensive scale.

that such illnesses are the consequence of inoculation of a scrofulous virus.¹ Moreover, a transmission of scrofula by vaccination from arm to arm is so much the less likely, that the vaccinifers are mostly in their first year, or at a time of life when the disease is not yet developed, or, if the phrase be preferred, is still latent.² I have already mentioned (p. 609) how this doctrine of inoculating with scrofula arose; in recent times it has been inflated to the most absurd extent by the anti-vaccinationists and turned to very good account for their purposes.

As regards clinical experience, then, there is nothing to warrant the opinion that scrofula is a contagious disease; and in that sense Begin³ long ago wrote, in somewhat incisive words: "Une ignorance et une crédulité stupides inventent la doctrine de la contagion des écouelles; une faculté de médecine⁴ donne sa sanction à cette opinion; trois ou quatre observateurs inhabiles croient avoir, dans les faits qu'ils ont recueillis, la confirmation de son exactitude; et bientôt la foule, copiste servile des opinions des autres, commente, amplifie et proclame enfin comme loi de la nature, ce que la plus simple observation infirme chaque jour."

Even if we take the other line of research, namely, the experimental, there has been, in my opinion, no evidence obtained in favour of the contagious character of scrofula. Kortum's indefensible experiments to infect children with the matter from scrofulous abscesses had no effect;⁵ nor had

¹ Lepelletier tells us that one of his colleagues had the hardihood to introduce a quantity of scrofulous pus into punctures made upon children on account of vaccination; the vaccine developed excellently, but of scrofulous illness there was not a trace (the incident is quoted by Phillips, l. c., p. 146).

² The prohibition in the vaccination law of Germany against taking vaccine lymph from the arm of a scrofulous child is in any case right; for, apart from the question here at issue, ailing persons in general are not suited to take vaccine matter from.

³ 'Dict. des Sc. méd.,' Paris, 1820, vol. 1, p. 293.

⁴ The reference is here to the declaration of the Paris faculty in 1758.

⁵ "Quippe materiem ex talibus (scrofulosis) ulceribus desumptam," says Kortum (p. 218), "puello sano ad latus colli integra cute infricavi, alio vero puero cuticula exiguo vulnuscule, velut in variolarum insitione fieri solet, disrupta, itidem in superiori colli regione, nempe infra et pone processum mastoideum applicavi, — et ne ullam quidem inde observare potui morbi communicationem." In justification of his experiment he adds: "Nemo dicat audacula hæec tentamina, cum firmissimis indubitatisque rationibus essem persuasus, nullum inde

those of Lepelletier, first upon guinea-pigs,¹ and afterwards upon himself. Scarcely had the parasitic doctrine raised its head, when Moretto² hazarded the conjecture that scrofula might be due to a vegetable or animal parasite, the grounds of his surmise being that "tuberculous growths" on animals and plants were caused by parasites, and that the heredity of scrofula presupposed a definite germ of disease. Subsequently Hüter³ developed the theory that the plasmatic vessels in childhood undergo dilatation owing to the large amount of the nutritive juices; and inasmuch as they extend to the surface layers of skin and mucous membrane, these latter lose that firmness which protects them from the invasion of inflammation-breeding organisms suspended in the air, so that the parasites enter and set up scrofulous inflammation, first in the skin and mucous membrane, and afterwards in the lymph-glands, which they reach by the lymphatic vessels. More recently Koch has adduced evidence that the tubercle-bacilli discovered by him are found also in scrofulous glands, and that the inoculation of the parasite (bred pure by cultivation) upon animals is followed by tuberculosis in them. I refrain for the present from going more particularly into the question of the causal relation of this parasite to the scrofulous process. When I come to speak of the connexion which subsists from the historical and etiological point of view *between pulmonary consumption and scrofula*, I shall have an opportunity of discussing that question. In the meantime I give it as my

damnum pueris subnasci posse. Imitentur Lectores, si placet, experimentula, eundemque eventum fore polliceor."

¹ He first shut up the animals in a small dark place, such as seemed to be particularly conducive to the development of scrofula; next he mixed their food with pus from typical cases of scrofula, in such quantities that each of them consumed half a teaspoonful daily. Also he injected eight to ten drops of the pus into the crural vein, and rubbed the matter into a wound made in the region of the inguinal lymphatic glands and into a smooth-clipped spot on the neck. The animals were killed a few months after, but in none of them was there any trace of scrofula. Lepelletier and his colleague Goodlad several times inoculated themselves with scrofulous pus, but no signs of scrofula ever followed (quoted in Phillips, p. 146).

² 'Annali univ. di medicina,' 1859, Settembre, p. 520.

³ 'Die scrophulose und ihre lokale Behandlung,' Leipzig, 1872 ('Volkmann's Sammlung klin. Vortr.,' No. 49).

opinion that the results arrived at hitherto by means of experimental research upon animals have not shaken the conviction of clinical experience that scrofula is non-contagious.

CHAPTER XVIII.

DIABETES.

§ 219. DEFECTIVE HISTORICAL RECORD.

The proved importance of the historical and geographical lines of pathological inquiry for the working out of the etiology of disease, or for elucidating the causal connexion of climatic, seasonal, telluric and social influences with the existence or spreading of maladies, is brought home to us with double force by the omissions and imperfections in our historical and topographical information about those diseases which stand most in need of some such etiological elucidation. For diabetes this holds good to the fullest extent as regards both the historical evidence and the topographical. The very manifest want of tolerably safe and exhaustive topographical details about that disease is explained by the fact that it is seldom seen at all; it has accordingly escaped the notice of all observers who were not specially on the outlook for it, most of all in those parts of the world where practitioners have had no opportunities of ascertaining the state of health of the population except within a very narrow circle; and in other countries the desideratum has been very inadequately supplied by the statistics of mortality, which are not only wanting in absolute trustworthiness so far as they go, but have assigned in only a trifling proportion of instances a special place to diabetes among the causes of death, ranging it in most cases under the general head of "chronic disorders of nutrition" or "diseases of the uropoietic system," and mixing it up with Bright's disease, gout, gravel and the like. It is obvious that hospital statistics cannot be depended upon in such an inquiry; for

diabetes is well known to be commonest among those classes who least resort to hospitals in their sickness. I have thought it well to premise this much as an apology for the sketchy character of what follows. It gives me the opportunity also of expressing the wish that subsequent investigators and statisticians may endeavour as far as possible to fill up this very perceptible lacuna.

§ 220. EARLIEST REFERENCES TO DIABETES.

The *history of diabetes* goes back to the most remote antiquity. The earliest accounts come from India; in the Ayur Veda of Susruta¹ we read: “Mellita urina laborantem quem medicus indicat, ille etiam incurabilis dictus est . .

. . Omnes urinales affectiones tempore incurabiles fiunt; ad mellitum urinae statum perveniunt et tunc insanabiles fiunt.”

In another passage² of that work, where the same disease is treated of circumstantially, the phrase occurs, “dulcis fit urina, sudor et phlegma;” and there can be no doubt that diabetes is the malady referred to.³ The occurrence of the disease in various parts of Europe and Asia during the later period of antiquity, and throughout the middle ages, is indicated in various ways in the writings of the Græco-Roman,⁴ Arabian and Arabistic schools; but from none of these does

¹ ‘Nidanasthana,’ cap. vi, in Hessler’s Latin translation, Erlangen, 1844, i, 184.

² ‘Chikitsitasthana,’ cap. xii, xiii, in the Latin transl., ii, 103.

³ Christie (‘Edin. Med. and Surg. Journ.,’ 1811, July, p. 285) gives an account of a Cingalese medical treatise of the fifteenth century of our era, translated from the Sanscrit, in which the disease is also referred to under the name of “madu mehe,” or “honey-urine.” It is a probable guess that the work in question may have been a translation of the treatise of Susruta.

⁴ There is nothing in the Hippocratic Collection which points to diabetes. The first mention of the disease is in Celsus (“Urina super potionum modum etiam sine dolore profluens, maciem et periculum facit,” lib. iv, cap. xx, § 2). The first to use the name of διαβήτης was Aretæus (‘De causis diuturn. morb.,’ lib. ii, cap. ii, and ‘De morbor. diuturn. curatione,’ lib. ii, cap. ii); he speaks of the malady as a mysterious one (θῶνμα πάθος) and of rare occurrence (οὐ κάρτα ξύνηθες ἀνθρώποισι), wherein the flesh and frame melt into urine (σαρκῶν καὶ μελέων ἐς οὖρον ξύνητης). Galen also mentions the disease in several passages of his writings (‘De symptom. differentiis,’ lib. i, cap. vi, Kühn’s ed., vii, 81; ‘De crisis,’ lib. i, cap. xii, Kühn’s ed., ix, 597); but he states in another

it appear that their authors had been acquainted with the sweet taste of the urine, and the same is true of the medical observers of the sixteenth century. Willis in his 'Pharmaceutice rationalis,'¹ published in 1674, calls attention to that property of the urine in diabetes; but it was not until a hundred years after that Dobson² showed that the taste of the urine depended on a sugary substance contained in it, which he detected by means of a fermentation experiment, and that the serum of the blood also in cases of diabetes had a sweet taste. Twenty years later, there was published the well-known essay on diabetes by Rollo,³ who was the first to discuss the subject thoroughly and who laid the foundation of all subsequent writings upon it.

§ 221. AREA OF PREVALENCE MOSTLY WITHIN THE TEMPERATE ZONE.

From the references of Indian, Græco-Roman and Arabian writers we obtain evidence that the geographical area of diabetes even in those remote times extended over a large part of *Southern Europe, Nearer Asia* and *India*. These data were afterwards supplemented by notices of the malady from almost every country of *Central* and *Northern Europe*; and towards the end of last century there came information of cases of diabetes among the European population of *North America*. In all those countries within recent times the

passage ('De locis affectis,' lib. vi, cap. iii, Kühn's ed., viii, 394) that he had seen only two cases of it. Short notices of it occur subsequently in the compendiums of all the later Greek physicians, as well as in Rhazes ('De re medica ad Alman-sorem,' lib. ix, cap. 78), and in Avicenna ('Canon,' lib. iii, Fen. 19, Tract. ii, cap. 17), and Avenzoar ('Alteisir,' lib. ii, Tract. ii, cap. vi, Venet., 1490, fol. 25); also in the Salernians, particularly Constantinus Africanus ('De morbis cognosc. et curand.,' lib. v, cap. 18, and 'Liber aureus,' cap. 39, § 2, Basil., 1536, 118, 182), and Platearius ('Practica,' "De aegritud. renum," cap. i, Lugd., 1525, fol. 219 b, or in de Renzi's 'Collectio Salernitana,' "De egritudinum curatione," Napoli, 1853, ii, 310), and in nearly all the writers of medical handbooks in the latter part of the middle ages.

¹ Sect. iv, cap. iii, Opp. Amstelod., 1682, p. 64.

² 'Med. Observ. and Inquiries by a Soc. of Phys.,' Lond., 1776, v, 298.

³ 'Account of two Cases of the Diabetes mellitus, &c.,' Lond., 1797 ('Notes of Diabetic Case,' Deptford, 1796).

disease has been seen in greater or less frequency; but whether it is more common in some of them than in others, it is impossible for the present to settle with any degree of probability except for a few places. In the following table I have put together the results of statistical returns for certain countries or cities; but I would attach no further importance to them than as proving that the disease, estimated by the mortality that it causes, is one of the rarest. It remains doubtful on what the considerable differences in the frequency of diabetes at various places depend; and whether such difference really exist, or are merely so brought out by the defectiveness of the returns.

Table of the death-rate from diabetes in various countries.

Locality.	Period.	Mean annual death-rate from diabetes.	Mortality from diabetes	
			per 1000 in-habitants.	per 1000 deaths.
England ¹	1852-69	550·0	0·027	1·25
Ireland ²	1841	118·0	0·014	0·74
Schleswig-Holstein ³	1871-79	14·3	0·014	0·65
Berlin ⁴	1877-79	30·0	0·029	0·94
Chemnitz ⁵	1871-74	2·5	0·035	1·00
Frankfort-on-Main ⁶	1865-80	3·4	0·035	1·60
Würzburg ⁷	1852-55	1·0	0·040	1·20
Brussels ⁸	1864-80	3·3	—	0·60
Philadelphia ⁹	1872-77	15·7	0·021	0·88

In *St. Petersburg* diabetes would appear to be very rare

¹ 'Thirty-second Annual Report of the Registrar-General, &c.,' Lond., 1871, 230.

² Wyld, 'Edinb. Med. and Surg. Journ.,' 1845, July 8.

³ Bockendahl, 'Jahresbericht der öffentl. Gesundheitsverh. der Provinz Schleswig-Holstein.'

⁴ 'Statistisches Jahrb. der Stadt Berlin.'

⁵ Flinzer, 'Mittheil. des statist. Bureaus der Stadt Chemnitz.'

⁶ 'Jahresberichte über die Verwaltung des Medicinalwesens der Stadt Frankfurt a. M.'

⁷ Virchow, in 'Verhandl. der Würzb. phys.-med. Gesellschaft,' x.

⁸ Janssens, 'Bull. de l'Acad. de méd. de Belgique,' Ann. 1865—1881.

⁹ 'Transact. of the Pennsylvania State Med. Soc.,' 1873—1878.

according to the corroborative statements of Attenhofer¹ and Lefèvre,² the former remarking that not a single case had occurred in his own practice or in that of a number of his colleagues during a period of six years. Otto³ gives the same account for *Copenhagen*, where there was no case of diabetes entered in the table of deaths from 1835 to 1838. There are references to diabetes in *Turkey* by Rigler,⁴ and in *Egypt* by Pruner⁵ and Griesinger;⁶ in *Morocco* also the malady is seen from time to time.⁷ Among the natives of *Senegambia*, on the other hand, Chassaniol⁸ tells us that he had not seen a single case; neither is there any mention of it in the records of English and French practitioners on the *Guinea Coast*. It is strikingly common in *Ceylon*,⁹ and at some points of India, particularly, as would seem, on the Coromandel Coast¹⁰ and in Bengal,¹¹ being much rarer in the Bombay Presidency.¹² From *China*, *Japan*, *Australia* and the *islands of the Pacific* there are no accounts of diabetes; neither is it mentioned at all by the authorities for *Central America* and the *West Indies*, while Blair¹³ explicitly says that in *Guiana* it is absolutely unknown. In *Vera Cruz (Mexico)*, Heinemann has seen it comparatively often.¹⁴ Jordaô's statement¹⁵ that it is not unfrequently met with in *Brazil* is directly contradicted by Jobim, who says that he

¹ 'Med. Topogr. der Hauptstadt St. Petersburg,' Zürich, 1817, 235.

² 'Lond. Med. Gaz.,' 1834, Novbr.

³ 'Transact. of the Prov. Med. and Surg. Assoc.,' 1839, vii, 237.

⁴ 'Die Türkei und deren Bewohner,' ii, 323.

⁵ 'Die Krankheiten des Orients,' 267.

⁶ 'Archiv für physiol. Heilkde.,' 1859, 5.

⁷ Ref. in 'Med. Times and Gaz.,' 1875, July, 96.

⁸ 'Arch. de méd. nav.,' 1865, Mai, 508.

⁹ Christie, l. c.; Davy, 'Account of the Interior of Ceylon,' Lond., 1821.

¹⁰ See Eyre, 'Madras Quart. Journ. of Med. Sc.,' 1860, Octbr., 341; Cornish, ib., 1861, July, 89; Ref. in 'Madras Monthl. Journ.,' 1870, May, 373; also the accounts by von Ruhde ('Bibl. for Laeger,' 1831, i, 281) for Tranquebar; Huillet ('Arch. de méd. nav.,' 1869, Févr., 83) for Pondicherry; and Auboeuf ('Contributions à l'étude de l'hyg. et des malad. dans l'Inde,' Par., 1882, 53) for Karikal.

¹¹ Eyre, l. c.

¹² Morehead, 'Clinical Researches on Disease in India,' Lond., 1856, ii, 297; Eyre, l. c.

¹³ 'Account of the last Yellow Fever Epidemic,' Lond., 1852, 20.

¹⁴ In 'Virchow's Archiv,' 1873, lviii, 178.

¹⁵ 'Considér. sur un cas de diabète,' Par., 1857.

did not observe a single case of it during a practice of many years at Rio ; and we may find a confirmation of this in the silence about the malady maintained by Sigaud, Rendu and other authorities on the diseases of that country. In *Peru* also, it would appear from Smith¹ and Tschudi,² to be extremely rare ; the latter, indeed, tells us that the Peruvian practitioners are quite unacquainted with it.

§ 222. ETIOLOGICAL QUESTIONS.

It is obvious that, from these scanty details of diabetes throughout the world, we can draw no just conclusion as to the influence of climate and the like on its geographical distribution. But the assertion often made, that a damp and cold climate disposes especially to the disease, is entirely upset by the facts adduced. The prevalence of the disease in Ceylon and some parts of India, particularly among high-caste Hindus whose *diet is exclusively vegetable* (Cornish), appears to favour the doctrine of Rhude, Davy and others, that its commonness has something to do with a diet that is mostly if not altogether non-nitrogenous. But the question would remain, why it is that diabetes is seldom or perhaps never seen in many other tropical countries of the Eastern and Western Hemispheres, in which the diet of the native population is the same.

¹ 'Edinb. Med. and Surg. Journ.,' 1841, Octbr., 400.

² 'Oest. med. Wochenschr.,' 1846, 473.

CHAPTER XIX.

GOUT.

§ 223. COPIOUS REFERENCES TO GOUT IN ANCIENT LITERATURE.

If our inquiry into the position of diabetes in place and time leads to no very satisfactory result, in consequence of the want of information about it in medico-topographical writings; the study of gout from the geographical and historical points of view meets with hardly less serious obstacles in the documents relating thereto, from the fact that the gouty process has often been confused with chronic rheumatism and with arthritis nodosa ("rheumatic gout") not only by the observers and authorities of former times but also by those of the modern period and of the latest date. The available writings upon gout possess, therefore, only a limited utility for the scientific handling of the malady in the particular directions of this work; more especially they are unserviceable in that much ventilated question whether the fluctuations in the number of cases, which would seem to have occurred from time to time according to contemporary data, had occurred really, and in particular whether the disease has undergone a considerable diminution of recent years as compared with last century; or whether such fluctuations are to be explained rather by the changing ideas of the day as to what was "gout"—by the varying connotation and scope of the term at various periods.

We are able with certainty to follow gout back as far as the time of Hippocrates, that is to say, the fifth century B.C.; there are many unambiguous references to *ποδάγρα* in the Hippocratic Collection, which show that the malady was

well known to the practitioners of that age.¹ It follows, moreover, from the quotations in the chapter on gout² in Caelius Aurelianus (that is to say, Soranus),³ that special attention had been given to gout by the post-Hippocratic Stoics (Diocles and Praxagoras) and by the Alexandrians (Herophilus and Erasistratus), whose writings have not come down to us, and that Erasistratus even wrote a monograph on the subject.⁴ In the Roman Empire of the West, gout appears to have been of frequent occurrence as early as the last years of the Republic;⁵ but all the writers on medicine of the time, as well as the poets and philosophers, are agreed in saying that it grew to be remarkably prevalent amidst the increasing luxury and sensuality under the early emperors. Neither does that prevalence of the disease appear to have been limited to Italy; for, according to the statement of Caelius Aurelianus,⁶ gout was reckoned among the maladies of common occurrence in Caria (south-western division of Asia Minor) and in Alexandria.

As early as the time of Pliny (first century of our era), a considerable increase in the number of cases had been remarked: "*Podagrae morbus*," says that writer,⁷ "*rarior solebat esse non modo patrum avorumque memoria, verum etiam nostra, peregrinus et ipse. Nam si Italiae fuisset antiquitus, latinum nomen invenisset.*" Seneca, the contemporary of Pliny, who, as a Stoic, missed no opportunity

¹ See 'Praenot. Coacae,' Sect. v, § 502, ed. Littré, v, 700; 'Aphorism.,' lib. v, § 25, and vi, § 28—30, 55; 'Epidem.,' lib. ii, sect. iii, § 12, e. c. v, 114; 'De affectionibus,' § 31, e. c. vi, 242; 'Prorrh.,' lib. ii, § 8, e. c. ix, 26. In the last mentioned treatise, which is probably later than Aristotle, the subject is treated with particular fulness.

² "*De morbis acutis et chronicis*," 'Morb. Chron.,' lib. v, cap. ii, Amstelod., 1755, p. 566.

³ It is well known that the treatise of Caelius Aurelianus is a translation, and in all probability a literal translation, of the work on medicine by the great methodist Soranus, who lived in Rome in the second century of our era.

⁴ It appears from a reference in Caelius Aurelianus that the King Ptolemy (probably Soter) was a sufferer from gout, and that Erasistratus treated him for it.

⁵ In evidence of this there are the exhaustive memoirs on the disease by Asclepiades and Themison, mentioned by Caelius Aurelianus. Cicero also mentions patients with the gout in many passages of his writings (e. g. 'Epistol.,' vii, 4, ad M. Mar., and 'De finibus,' v, 3).

⁶ L. c., p. 558.

⁷ 'Hist. nat.,' lib. xxvi, cap. lxiv, ed. Franzius, Lips., 1788, vii, 851.

in his writings of animadverting on the dissolute and riotous life of Rome and on the after-consequences to the health of the community, dwells upon the fact that podagra was common even among the women; and he adds the remark that it was not surprising, for their sex emulated the excesses of the men in every respect.¹ "In the time of Hippocrates," says Galen,² "there were only a few who suffered from podagra, such was the moderation in living (διὰ τὸ τοῦ βίου κόσμιον); but in our own times, when sensuality has touched the highest conceivable point, the number of patients with the gout has grown to an extent that cannot be estimated (ἄπειρόν τι τὸ πλῆθος τῶν ποδαγριῶντων)." The same enormous prevalence of the malady afforded a subject to the satirist Lucian for his little comedy of "Τραγαποδάγρα," in which he introduces Podagra as a goddess and represents her in a witty fashion as exercising absolute sway over mankind.³ The most prominent physicians of the time took occasion also to write on the subject at great length, the chief treatises that have come down to us being those of Aretaeus⁴ and Caelius Aurelianus (that is to say, Soranus).

The word *ἀρθρίτις*, used by Galen, is applied by him and the later Greek and Roman writers on medicine to express "inflammation of the joints" in general; while of special forms of it, good descriptions are given of *ισχιάς* or inflammation of the hip-joint, of *ποδάγρα* and *χειράγρα* (distinguished as *νοσήματα θερμὰ καὶ ψυχρά* according to a humoral principle founded on the morbid phenomena), and lastly of gouty

¹ 'Epistol.,' 95, Opp., ed. Haase Lips., 1853, iii, 302: "Non mutata feminarum natura, sed vita est; nam cum virorum licentiam aequaverint, corporum quoque virilium incommoda aequarunt. Non minus pervigilant, non minus potant, et oleo et mero viros provocant; aequae invitis ingesta visceribus per os reddunt et vinum omne vomitu remetiuntur." Seneca mentions and treats of gout in many other passages of his writings (e.g. 'Epist.,' 53, 67, 78, ed. cit., iii, pp. 111, 152, 197); and in his 'Lud. de morte Claudii Caesaris' (§ 13, ed. cit., i, 273), he remarks that the Emperor Claudius was a sufferer from it.

² 'Comment. in Hipp. aphorism.,' cap. xxviii, ed. Kühn, xviii, A. 42. Other references to gout in the writings of Galen will be found in the following: 'De sanitate tuenda,' lib. vi, cap. vii, e. c. vi, 415; 'Method. med.,' lib. vii, cap. xi e. c. x, 513; 'De compositione medic. secund. locos,' lib. x, cap. ii, e. c. xiii, 331; 'De remed. parabil.,' lib. i, cap. xvi, e. c. xiv, 383; 'De theriaca lib. ad Pisonem,' cap. xv, e. c. xiv, 274.

³ The little comedy of *Ωκύπους* appended to this drama, and dealing with the same subject, is apocryphal.

⁴ 'De causis et signis diuturnor. morbor.,' lib. ii, cap. xii.

nodules under the name of *πόροι*, or swellings of stony hardness.¹ We find mention already of the metastases of gout also, Galen speaking of gout flying to the stomach (*μετέστη εἰς τὴν γαστέρα*),² and to the lungs,³ and Aretaeus of gouty asthma.⁴ It can hardly be doubted that chronic articular rheumatism, and particularly arthritis nodosa, had entered into their conception of "podagra."

Whether any diminution of the number of cases occurred in the time of the later Empire and in the middle ages is not ascertainable from the medical writings of those periods; the less so that "arthritis" (or as the mediæval writers, and, so far as I know, Constantinus Africanus first, name it "arthetis") would appear to have been more widely prevalent than formerly, while the term "podagra" occurs less often (the disease being now and then called "gutta" or "drop"⁵), and in the end becomes merged almost entirely with other diseases of the joints under the general name of "arthritis." The very prolix dissertations on gout in the compendiums of Aetius,⁶ Paulus Ægineta,⁷ and Alexander of Tralles⁸ (who occupies himself almost entirely, and at enormous length, with directions for treatment), as well as in the writings of the Arabians, particularly Serapion the elder,⁹ Rhazes,¹⁰ Abulcasim,¹¹ Avicenna,¹² and Avenzoar¹³ (whose work is one of the best), merely reproduce the data of the earlier Greek practitioners, and give us no means of forming a conclusion as to the frequency of the disease. The same is true of the works of mediæval physicians of the West, of Constantinus Africa-

¹ The word *πόροι* is also used to designate bony callus.

² 'Method. med.,' l. c.

³ 'De theriaca lib. ad Pisonem,' l. c.

⁴ L. c., ed. Kühn, p. 174.

⁵ The first use of the term "gutta" that I can find is in Valescus de Tharanta, who defined it: "Est passio in ligamentis et nervis juncturarum ex humore vel ventositate ad eos decurrentibus a membris superioribus vel circumvicinis." It is here used, therefore, in the same sense as "gutta in oculis" applied to cataract. It is clear that the French "goutte" and the English "gout" are derived from "gutta."

⁶ 'Sermo,' xii, cap. vi—xlviii.

⁷ Lib. iii, cap. 78.

⁸ Lib. xii.

⁹ 'Practica Tract.,' iv, cap. 23—30, Lugd., 1525, fol. 44, *seq.*

¹⁰ 'De re medica,' lib. ix, cap. 90, and 'Lib. de affect. juncturarum.'

¹¹ 'Method. med.,' lib. i, cap. 45.

¹² 'Canon,' lib. iii, Fen. 22, tract. 2, cap. 55, *seq.*

¹³ 'Theisir,' lib. ii, tract. vii, cap. 30, Venet., 1490, fol. 33.

nus,¹ and of the Salernians Platearius,² Valescus de Tharanta,³ Arnaldus Villanovanus,⁴ Savonarola,⁵ and Guainerio;⁶ as well as of the monograph on the disease by Demetrius Pepagomenos,⁷ a physician of the thirteenth century practising at the Byzantine court, which is for the most part based upon the works of Paulus and Alexander.

The literature of gout in the sixteenth century opens with the writings of Paracelsus⁸ on podagra (of very doubtful genuineness, however), which have no historical interest except that they are the first to introduce the German name of "Zipperley," applied to the disease, and that the discussion of the subject in them is elaborate enough to prove to us that gout must have played a very prominent part among the diseases of the time. The same conclusion is justified also by the numerous treatises on podagra in the medical literature of the sixteenth, seventeenth, and eighteenth centuries; although we are still prevented, by the confounding of true gout with chronic rheumatism and arthritis nodosa, from coming to any definite opinion as to the amount of the disease. According to these writings, however, it was spread over the whole of Europe.

A new source of error arose after Sydenham's classical description⁹ of the malady as he observed it in his own person, and after Hoffmann's¹⁰ and van Swieten's¹¹ excellent works on

¹ 'De morb. cognosc. et curand.' lib. vi, cap. xix, Basil., 1536, 137; and 'Lib. aureus,' cap. xlv, e. c. 185.

² 'Practica,' 184 b, and in de Renzi, 'Collect. Salernit.,' ii, 349—356.

³ 'Philonium,' lib. vi, cap. 23, Lugd., 1490, fol. 295 b.

⁴ 'Parabolae medicationis,' Opp., Basil., 1585, 985, and 'Breviar.,' Lib. ii, cap. 45, e. c. 129 h.

⁵ 'Practica tract.,' vi, cap. xxii, Rubr. x, Venet., 1497, fol. 272 a.

⁶ 'Commentar. de aegritud. junctur.,' cap. i, *seq.*, in 'Practica,' Lugd., 1534, fol. 171 b (contains a few observations by the author himself).

⁷ 'De podagra libellus,' Romae, 1517 (reprint in Stephan's collection, Paris, 1567, p. 837).

⁸ 'Buch von den tartarischen Krankheiten,' cap. 19, Opp. Strassb., 1603, 313; 'Vom Podagra,' e. c. 539; 'Liber de podagricis' (in German), e. c. 563.

⁹ 'Tract. de podagra,' Opp., Genev., 1736, i, 300.

¹⁰ 'Med. ration. system.,' Tom. iv, Sect. ii, cap. xi, Opp., Genev., 1753, ii, 399; 'Diss. de genuino dolor. podagr. remed.,' Hal., 1697, Opp. Supp., ii, Pars. ii, 173; 'Diss. de podagra retrocedente in corpus,' Hal., 1700, Opp., ib., 187; 'Diss. de cura doloris podagr.,' Hal., 1738, Opp., ib., 180.

¹¹ 'Comment. in Boerhaave aphorismos,' §. 1254—1282, Lugd. Batav., 1764, iv, 287—393.

the subject, which drew the line sharply between gout and rheumatism. Coincidentally with the more precise determination of the existence of a gouty diathesis, or, in other words, with the proof of a constitutional character for the disease, the province of "gouty ailments" underwent an extension beyond all bounds; so that all the advantage on the one side by the sharper separation of gouty affection of the joints from other degenerative articular changes (rheumatism in particular), was lost on the other side by overstraining the doctrine of "internal gout," which was resorted to, along with the "hæmorrhoidal disease," whenever there was a difficulty about the diagnosis. It is only within recent times, and mostly since morbid anatomy began to be cultivated, that sobriety of view has entered into the doctrine of gout; and accordingly the large place that the disease held in former centuries has been a good deal restricted, although it may still happen to us at the present day to hear and read of rheumatic arthritis and arthritic rheumatism.

Care is needed, therefore, in estimating and weighing the data as to gout, so as to arrive at its history in the past and its prevalence in the present; and we may take it as certain that its comparative rarity in recent times beside former centuries depends not a little upon the more precise diagnosis of the malady, or upon the disentanglement of what is included under "gout" from among other forms of disease resembling it. At the same time it cannot be doubted, after the trustworthy observations¹ that have been made in Spain, Italy, Belgium, Holland, Switzerland, and even on the classical soil of England, that gout in recent times has undergone a real and very considerable abatement compared with previous centuries, just as in the early period of the Roman Empire it underwent a considerable increase as compared with the earlier age of antiquity. So far as we may conclude from the medico-topographical records before us, there are very few parts of the world where it counts at the present day among the more common constitutional disorders of nutrition, and there are none where it has the character of a truly endemic malady.

¹ See in particular the admirable research of Corradi, '*Della odierna diminuzione della podagra e delle sue cause*,' Bologna, 1860; also the writings of Dolleman

§ 224. PRESENT GEOGRAPHICAL DISTRIBUTION : RARE
IN THE TROPICS.

The *geographical area of gout* at the present time includes a very large part of the temperate zone ; but within that area there are very considerable differences obtaining in the amount of the disease at the various points of observation, for which there is no arithmetical expression to be had. In *Spain*, according to somewhat antiquated information,¹ it would appear to be particularly common in Asturias. In *Italy* the regions most affected were the Alpine valleys of Piedmont,² Naples³ (among the well-to-do) and Sardinia.⁴ For *France*, Lorraine and Normandy⁵ are given as the chief seats ; in the large towns of the Department of the Rhone, also, it is far from rare even among the less comfortable classes of the populace, one of the authorities⁶ remarking that “ il suffit d’avoir exercé dans une grande ville, pour savoir que dans les classes inférieures on rencontre un assez grand nombre de gouteux.”

For *Switzerland* we hear from Lebert⁷ that gout has grown rarer among the upper classes “since manufactures and railways have begun to claim many of their energies formerly unused.” In *Belgium*, where the disease has become a good deal less common,⁸ it is now met with mostly in Flanders and in the Walloon provinces.⁹ In *Holland* also it is now seen on the whole rarely.¹⁰ For *Germany* we have accounts for Holland, Coley for Belgium, Lebert for Switzerland, and Owen, Fuller, Budd, and others (see p. 655) for England.

¹ Thiéry, ‘Observ. de physique et de médecine faites en . . Espagne,’ Par., 1791, ii, 108.

² ‘Brunner, ‘Verhandl. der Schweiz. ärztl. Gesellsch.,’ 1829, i, 151.

³ de Renzi, ‘Topogr. e statist. med. della città di Napoli, &c.,’ Nap., 1845, 326.

⁴ Moris in de la Marmora’s ‘Voyage.’

⁵ Simonin, ‘Rech. topogr. et méd. sur Nancy,’ Nancy, 1854, 250; Charcot, ‘Med. Times and Gaz.,’ 1867, March, 245.

⁶ Marmy et Quesnois, ‘Topogr. stat. et méd. du Depart. du Rhône, &c.,’ Lyon, 1866, 548.

⁷ ‘Handbuch der pract. Med.,’ 1859, ii, 898.

⁸ Coley, ‘Remarks on the Climate and the Diseases occurring in Belgium,’ Bruss., 1852, 163.

⁹ Meyne, ‘Topogr. méd. de la Belgique,’ Brux., 1863, 211.

¹⁰ Dolleman, ‘Disquis. de plerisque apud Belgas septentrionales endemiis morbis,’ Amstelod., 1824, 55.

of its comparatively frequent occurrence in Hamburg,¹ Mecklenburg,² the Harz,³ Bremen,⁴ Göttingen,⁵ Dresden,⁶ Wiesbaden,⁷ Ludwigsburg,⁸ Passau,⁹ and Upper Austria;¹⁰ in Berlin it came under Traube's notice extremely seldom.¹¹ *England* still remains one of its principal seats, although there too, as we learn from Fuller, Budd,¹² Watson,¹³ Forbes,¹⁴ and others, there has been a considerable decrease noticeable. Forbes writes: "Two of the oldest practitioners in the district (the Land's End), each resident in a small country town, assured me that in their earlier practice, that is, forty or fifty years before, gout was much more frequent than at present—in the proportion, they said, as a hundred to one." It is very remarkable, on the other hand, that *Scotland* and *Ireland* have always enjoyed a notable immunity from gout.¹⁵ From *Denmark* I know of only one account, by Otto,¹⁶ who says that the malady is relatively common, especially in Copenhagen. For *Sweden* and *Norway* there is no recent information about it. In *Lapland* (according to Linnæus), and in *Iceland* and the *Farøe Islands*¹⁷ it is quite unknown. In *Russia*, if we may still depend upon accounts dating from the earlier years of the century, gout is of frequent occurrence in the Baltic provinces¹⁸ and in St. Petersburg;¹⁹ it is

¹ 'Hamburg in naturhistor. und med. Beziehung,' Hamb., 1830, 89.

² Ebstein, 'Die Natur und Behandl. der Gicht,' Wiesbad., 1882, 138.

³ Id.

⁴ Heineken, 'Die freie Hansestadt Bremen, &c.,' Brem., 1837, ii.

⁵ Ebstein, l. c.

⁶ Mayer, 'Versuch einer med. Topogr. von Dresden', Stollberg, 1840, 288.

⁷ Müller, 'Med. Topogr. der Stadt Wiesbaden', Wiesb., 1846.

⁸ Höring, 'Württemb. med. Correspondenzbl.', 1839, ix, 275.

⁹ Friedrich, 'Bayr. ärztl. Intelligenzbl.', 1855, 353.

¹⁰ Gugger, 'Oest. med. Wochenschr.', 1843, 785.

¹¹ 'Berl. klin. Wochenschr.', 1865, 474.

¹² In Tweedie's 'Library of Medicine,' v, 208.

¹³ 'Lancet,' 1842, Nov.

¹⁴ 'Transact. of the Provincial Med. and Surg. Assoc.,' 1839, iv, 203.

¹⁵ Scudamore, 'Treatise on the Gout,' Germ. ed., Halle, 1819, 54. In Glasgow gout is very rare, even among the rich classes.

¹⁶ 'Transact. of the Prov. Med. and Surg. Assoc.,' l. c.

¹⁷ Manicus, 'Bibl. for Laeger,' 1824, i, 15.

¹⁸ Bluhm, 'Beschreibung der in Reval herrschenden Krankheiten,' Marb., 1790, 141; Moritz, 'Spec. topogr.-med. Dorpat.,' Dorp., 1823.

¹⁹ Attenhofer, l. c., 231.

also reported from Odessa.¹ On the other hand it is exceedingly rare to meet with it in the governments of Samara² and Kasan,³ and in Transcaucasia it would appear to be quite unknown.⁴ In Turkey, also, cases of gout are rarely seen.⁵

In the tropical and subtropical regions of *Asia* gout either does not occur at all or only in exceptional cases. This holds good for *Syria*, *Persia*, and *Arabia*,⁶ although in those countries, to judge by the writings of Arabian and Syrian physicians of the middle ages, gout was by no means a rarity formerly. With reference to gout in *India*, Ainslie, who had thirty years' experience, says: "I do not think that I ever knew but one Hindoo who had a well-marked gout; the Mahometans are not so fortunate in this respect. Those Europeans who are subject to the attacks of it have, for the most part, long intervals betwixt the fits, and when they do come they are generally slight."

On the Himalayan slope it would appear that the disease is not altogether rare⁷ (whether among natives or Europeans is not mentioned); and in the *East Indies* it is given as a not uncommon malady even among the natives.⁸ On the other hand it is quite unknown in *Ceylon*,⁹ or among the natives of *Assam*.¹⁰ For *China* there is only one notice before me, relating to Amoy,¹¹ where the disease has been seen rather often among the indigenous population. In the medico-topographical records of the *Australian continent* and the *islands of the Pacific* there is not a word said of the occurrence of gout. According to the account of Thomson,¹² dating from

¹ Andrejewsky in 'Gräfe and Walther's Journal,' 1833, xx, 277.

² Ucke, 'Das Klima und die Krankheiten der Stadt Samara,' Berl., 1863, 211.

³ Erdmann, 'Med. Topogr. des Gouv. Kasan,' Riga, 1822, 154.

⁴ Ref. in Hecker's 'Annal für wissensch. Heilkde.,' 1833, xxxi, 331.

⁵ Oppenheim, 'Ueber den Zustand der Heilkunde . . in der Türkei,' Hamb., 1833, 76; Rigler, 'Die Türkei und deren Bewohner, &c.,' ii, 365.

⁶ Marshall, 'Edinb. Med. and Surg. Journ.,' 1832, Oct., 347; Tobler, 'Beitr. zur med. Topogr. von Jerusalem,' Berl., 1855, 41.

⁷ Farquhar, 'Indian Annals of Med. Sc.,' 1863, April, 464.

⁸ Heymann, 'Krankh. der Tropenländer,' 181; van Leent, 'Arch. de méd. nav.,' 1867, Oct., 246.

⁹ Davy, 'Account of the Interior of Ceylon.'

¹⁰ Beaufile, 'Arch. de méd. nav.,' 1882, April, 266.

¹¹ Friedel, 'Beiträge, &c.,' 109; Ref. in 'Arch. de méd. nav.,' 1866, Sptbr., 166

¹² 'Brit. and For. Med.-Chir. Rev.,' 1855, April.

1837, the disease had never been seen in New Zealand; nor in the Hawaiian Islands according to Chapin¹ (1855).

The *African continent*, also, with the islands adjoining it, enjoys an almost complete immunity from gout. That is the expressed opinion of all the authorities for *Egypt*,² *the countries of the Niger*,³ *Algiers*,⁴ *Senegambia*,⁵ *the West Coast*,⁶ and *Madeira*.⁷ The only exceptions to this are found in Vinson's notice⁸ (somewhat untrustworthy) of its common occurrence among the Hovas occupying the plateau of *Madagascar*, and in the statement of Ferrini⁹ (also questionable) that it is not unfrequently seen in *Tunis*.

In the *Western Hemisphere*, so far as one may judge from the information about gout that comes to us thence, the malady is for the most part confined to countries within the temperate latitudes. In *Greenland* it is extremely rare,¹⁰ and there is no mention of it at all for Canada and other territories in the far north. In the large and populous towns of the *United States*, supplied with the luxuries of Europe, gout would appear to be as common as it is under the same circumstances in the Old World,¹¹ although our information on the subject is scanty. According to a notice of the year 1830 by Hildreth,¹² for Washington, gout was quite unknown among the indigenous inhabitants of that region. In Vera Cruz, Heinemann¹³ saw only two cases of it during six years. There is no mention of it in the medical accounts from Central America. In the *West Indies*, as we learn from both early and recent authorities,¹⁴ it is seen in rare cases only. The

¹ 'Amer. Journ. of Med. Sc.,' 1837, May, 93.

² Röser, 'Ueber einige Krankheiten des Orients,' Augsb., 1837, 73; Clot-Bey, 'Aperçu gén. sur l'Egypte,' ii, 319, u. a.

³ Brocchi, 'Giornale,' v, 559.

⁴ Bertherand, 'Méd. et hyg. des Arabes,' Par., 1855.

⁵ Chassaniol, 'Arch. de méd. nav.,' 1865, Mai, 507.

⁶ Copland, 'Dictionary,' iv.

⁷ Kämpfer, 'Hamb. Zeitschr. für Med.,' 1847, xxxiv, 159.

⁸ 'Gaz. hebdom. de méd.,' 1866, Nr. 49, Feuille.

⁹ 'Saggio sul clima e sulle precipue malattie della città di Tunisi, &c.,' Milano, 1860, 238.

¹⁰ 'Bemaerkn. om Gronlands Sygdomsforhold,' Kjobenh., 1864, 30.

¹¹ See Hosack, 'Essays,' New York, 1824, ii, 233.

¹² 'Amer. Journ. of Med. Sc.,' 1830, Febr., 330.

¹³ In 'Virchow's Arch.,' 1873, lviii, 161.

¹⁴ Dancer, 'History of the late Expedition against Fort St. Juan, &c.,' Lond.,

same is true for *Guiana*; ¹ and still more so for *Brazil* where gout is almost unknown, according to the unanimous opinion.² It applies also to *Peru*, where Smith³ saw only one case during a residence of more than ten years; but it would appear to be more common among the creoles of the Sierra, and in *Chili* also it occurs not unfrequently.⁵

§ 225. HIGH DEGREE OF HEREDITY.

It may be said to be a doctrine generally admitted at the present day, and beyond all questioning, that the process of gout depends upon a constitutional disorder of nutrition, that a morbid diathesis—the uric-acid diathesis—underlies it, and that the diathesis is sometimes inherited and sometimes acquired. The task of historical and geographical research, accordingly, is to inquire what are those factors, involved in circumstances of climate, manner of life, and of nationality, and associated with the distribution and degree of prevalence of the malady in place and time, which may be brought into causal connexion with the pathogenesis; or, otherwise expressed, it is to explain the influences upon which the development of the gouty diathesis depends, or which seem calculated to further the evolution of the disease itself where the diathesis had been either inherited or acquired.

The *heredity of gout* (or of the gouty diathesis) was a subject about which the physicians of antiquity and the middle ages were perfectly agreed. Galen lays special

1781; Lemprière, 'Pract. Observ. on Diseases . . in Jamaica,' Lond., 1799, i, 50; Forström, 'Svensk. Läk. Sällsk. Handl.,' 1817, iv, 231; Chassaniol, 'Arch. de méd. nav.,' 1865, Mai, 507; Ruzf (ib., 1869, Novbr., 350) saw twenty-eight cases of gout in Martinique during a long series of years.

¹ Rodschied, 'Bemerkungen u. s. w.,' 172. The statement of Blair ('Account of the last Yellow Fever Epidemic, &c.), that gout is of common occurrence in British Guiana, along with rheumatism, is most probably based on a laxity of diagnosis.

² Martius, 'Das Naturell und die Krankheiten der Urbewohner Brasiliens,' Münch., 89; Dundas, 'Sketches of Brasil,' Lond., 1852, 37; Sigaud does not mention it at all.

³ 'Edinb. Med. and Surg. Journ.,' 1841, Oct., 399.

⁴ Tschudi, 'Oest. med. Wochenschr.,' 1846, p. 731.

⁵ Ref. in 'Arch. de méd. nav.,' 1864, Août, p. 107.

stress on that factor in his explanation of the steadily increasing spread of the disease in the Roman Empire;¹ Aetius says, in fact, that the development of the malady is in most cases to be referred to hereditary transmission;² while, in modern times, Cullen,³ Hamilton,⁴ and others, go so far as to take the inheritance of the diathesis as the sole determining cause of the disease. Although the last of these opinions is decidedly an exaggeration, yet heredity is a factor in the etiology that is to be rated very highly. Scudamore⁵ was able to trace heredity in 34 cases out of 77, Patissier⁶ in 34 cases out of 80, Gairdner⁷ in 140 cases out of 156. Of 65 cases of gout that came under his treatment at a mineral spa, Braun⁸ could not find one in which there was not some evidence of a disposition inherited from the parents or grandparents. Garrod⁹ found that the half of all his hospital cases of gout could be referred to heredity; while his experience in private practice leads him to estimate the cases of inherited gout at 75 per cent. of the whole. An interesting case given by him is that of a gouty patient, fifty years of age, who stated that the disease had been handed down in his family from father to son for 400 years. But, however highly we may rate this factor in the etiology, we learn from the history of the disease, from the fluctuations in its amount from period to period, and above all from the very considerable abatement of the malady in recent times (a fact which cannot be doubted), that even the here-

¹ In 'Comment. in Hipp. Aphor.,' l. c., where his words are: "*προσελήλυθε δὲ ταῖς εἰρημέναις αἰτίαις, δι' ἃς νῦν ποδαгриῶσι πολλοί, καὶ τὸ πατέρων τοὺς πλείστους γεγονέναι καὶ πάππων ἤδη ποδαгриκῶν, ἐφ' ὧν δηλονότι τὸ σπέρμα μοχθηρότερον ἦν.*"

² L. c., cap. vii, e. c. 309: "Ut plurimum vero aptitudines a parentibus in filios ac posteros transferuntur."

³ 'First Lines in the Practice of Physic,' Germ. ed., i, 12, Leipz., 1778, i, 289.

⁴ 'Letters on the Cause and Treatment of the Gout, &c.,' Lynn (Norfolk), 1809.

⁵ L. c., 40. This is the statement in the translation which I have used. Garrod quotes Scudamore as giving 523 cases, of which 309 were proved to be hereditary. I suppose these are the figures in the 4th edition of Scudamore's treatise (1822), which I have not the means of referring to.

⁶ 'Report of the Paris Academy.'

⁷ 'On Gout,' Germ. transl. by Braun, Wiesb., 1858.

⁸ 'Beiträge zu einer Monogr. der Gicht,' Wiesb., 1860, 53.

⁹ 'Treatise on Gout and Rheumatic Gout,' Lond., 1876, 209.

ditary diathesis stands in a certain dependence on exterior influences, that it becomes aggravated or weakened under those influences, and that in some circumstances it may even be altogether abrogated.

§ 226. CONNEXION WITH LUXURIOUS LIVING ; EXCEPTIONS BOTH POSITIVE AND NEGATIVE.

Among such exterior influences, apparently capable not only of heightening the disposition towards the disease and of furthering its actual development, but even of evoking the gouty diathesis itself and so of inducing gout apart from heredity, the individual's manner of dieting and of living takes the first place. All medical observers, who have gone at all closely into the causes of gout, are agreed that the malady is commonest by far among the rich or better-off classes of society, being met with to a much smaller extent among the working classes and the poor ; and that a voluptuous or luxurious table, implying the unstinted use of spirituous liquors and animal food, especially when associated with want of exercise, if perhaps not absolutely a *conditio sine quâ non* in the pathogenesis, is at any rate the chief occasion of the gouty diathesis being established, or, where it had existed previously, of its being aggravated to the point of the evolution of the actual disease.

As early a writer as Galen, who agrees in this with his non-professional contemporaries, had said : “κατὰ μὲν τοὺς Ἱπποκράτους χρόνους ὀλίγοι παντάπασιν ἐποδαγρίων, διὰ τὸ τοῦ βίου κόσμιον, ἡνξημένης δὲ τῆς τροφῆς εἰς τοσοῦτον ἐν τοῖς καθ' ἡμᾶς χρόνοις, ὥς ἂν μὴδ' ἐπινοεῖν ἔστι προσθήκη αὐτῇ, ἀπερὸν τε τὰ πλῆθος τῶν ποδαγριῶντων ἐστίν.”

Among the principal causes of gout Aetius reckons “ebrietates,” and “consuetorum exercitiorum intermissiones ;” and the opinions of all subsequent Greek and Arabian writers on medicine are to the same effect. “Plurimum innascitur haec passio,” says Constantinus, “suaviter et quiete viventibus, et exercitia negligentibus et purgationes et corporis mundificationes nolentibus, maxime cum multum comodant atque bibant.” Arnaldus has a similar explanation, and he adds these words : “Fiunt autem in praelatis et in his qui fuerunt pauperes et postea ad divitias et prosperitates ascenderunt.”

¹ ‘Comment. in Hipp. Aphor.,’ l. c.

We shall hardly err if, following the unanimous testimony of contemporary writers, we connect the increase in the number of cases of gout and the enormous diffusion that it reached in the early period of the Roman Empire with the luxuriousness of the Roman life, which rose at that time to the most riotous excesses ; and pointing in the same direction is the decline in the number of cases in modern and recent times under the influence of rational dieting. It is also worthy of all attention in our inquiry into the importance of that factor, that in the tropics, where gout is rare in general, only those circles of society are affected by it who cultivate the pleasures of the table in contrast to the sober-living natives. Thus we learn from Rufz that the few cases of gout which he had in Martinique were exclusively among rich people. It is observed by Chassaniol that cases of gout occur from time to time in negroes in the West Indies, but only among the moneyed class of them. In India, as we have seen, it is met with only among Europeans and Mohammedans, and never among the temperate-living Hindus. In Egypt also it is not found except in Europeans and Turks who give themselves up to a life of luxury.

This factor, we repeat, is a most important one in the production of the gouty diathesis or of an actual attack of gout ; but we cannot forget, on the other hand, that there are not unfrequently attacks of gout quite independently of inherited diathesis, in persons who had been in no wise subject to those harmful influences, but, on the contrary, had kept a very simple and even meagre table. Thus Friedrich, in Passau and its vicinity, found cases of gout among people living in a very moderate way ; Dickson tells us that gout is unusually common among the Custom-House officers in London, but that it occurs in them altogether irrespective of their manner of life ; and Marmy speaks of the comparatively numerous cases of gout among the poorer classes in Lyons. On the other hand, daily experience teaches us that large numbers of people who are given to the immoderate use of alcohol (wine or beer), whose diet is mostly animal food, and who lead the most luxurious kind of life, continue quite free from gout. This fact confronts us on the greatest scale in the tropics, where the well-to-do classes are extremely

seldom affected with gout, notwithstanding errors in their diet of every kind. On this point Dundas is very decided, on the strength of his observations in Brazil.¹ Curiously enough his observations have received little attention in the writings upon gout, even in the works by his own countrymen.

The exemption, he says, which the natives of Brazil, and almost equally the foreign residents, enjoy from gout is all the more remarkable that the prevailing usages and practices in the higher classes of native society, as well as among the well-to-do foreigners, would rather lead us to expect gout to be of necessity a very frequent occurrence. The life led by the rich classes of society is almost without exception an inactive and indolent one. There is no exercise for their mental and bodily powers, and for that reason gross sensuality is all the more indulged in; and although the Brazilians are not exactly given to excesses in wine-drinking, they are fond of frequent and plentiful meals of animal food and of having their dishes highly flavoured. Here, accordingly, we have in unusual perfection all those conditions which are looked upon in Europe as the most material causal factors in the development of the gouty diathesis. But gout does not occur in Brazil; or, if it do occur at all, it is extremely rare. . . . The immunity from gout enjoyed by the residents in this hot climate is a thoroughly well-established fact; but it will follow conclusively from the sketch just given of the manner of life of the Brazilians that there is no ground whatsoever for explaining that exemption by any sober mode of living which they practise, or by a diet that is poor in nitrogenous elements in comparison with the diet of higher latitudes. And the same may be proved for the inhabitants of other tropical regions, and particularly for those of other countries in South America. We should thus have to seek an explanation of the phenomenon among circumstances of a different kind.

I shall not go farther into the theory which Dundas works out in connexion with the above. I merely draw from his observations, taken along with facts already adduced, the following conclusions: that the development of the gouty diathesis (irrespective of heredity) is undoubtedly under the influence of an erroneous manner of living and feeding; that it is not an effect, however, due to the sum of all those harmful things previously enumerated,—to excesses, or to an intemperate and luxurious manner of life in general; but that in the manner of life, there must be some definite, and in a measure specifically active factor to constitute the true

¹ 'Sketches of Brazil, including New Views of Tropical and European Fever, Lond., 1852.

cause of the malady, a factor which may make itself felt irrespective of gross errors of diet, or in those who lead a temperate life; and that this factor is one which may be overpowered and rendered harmless to the organism when other influences are brought into play or some particular state of body is induced. Of the nature of that specific cause of the gout, neither anatomy, nor clinical observation, nor etiology has told us anything as yet.

§ 227. INFLUENCE OF SEASON AND CLIMATE.

In the course of this chapter it has been several times mentioned, and it will be apparent also from the sketch of the geographical distribution, that gout is of rare occurrence in low latitudes. Hereupon arises the question whether this relative immunity of the tropics depends on the *climate*; or, in other words, what influence upon the occurrence and diffusion of the disease the *kind of weather* can be shown to exert.

In the very earliest writings on gout, such as those of Hippocrates, Galen, and Caelius Aurelianus, as well as in those of nearly all the later observers, such as Sydenham, van Swieten, Scudamore, Dickson, Garrod and others, spring and autumn are given as the proper seasons of gout. The general view is that damp and cold weather are specially apt to bring on paroxysms of the malady; and Garrod in particular says that even in chronic cases of gout the patients are usually much better during the summer than in other seasons of the year. The temperature and moisture of the air accordingly exert an undoubted influence on the course of the malady; and it is reasonable to suppose that weather-conditions of a favorable kind, such as the rather high and steady temperature peculiar to the tropics, would not be without significance even for the existence or non-existence of the disease in itself, and that the rare occurrence of gout in low latitudes is partly at least due to the influence of the climate. The opinion that has been several times put forward, to the effect that the immunity from gout enjoyed by residents in the tropics is referable solely to their temperate

and abstemious manner of life, can really apply to only a part of the native population; for the Europeans and the well-to-do classes of the other nationalities, it is by no means relevant, as Dundas has shown for Brazil, and as one might prove in like manner for India. We shall have to withhold our explanation of why the tropical climate is adverse to the development of the disease or conducive to a mild type, until such time as we have obtained a better insight into the real nature of the gouty diathesis, and the etiological factor underlying it.¹

§ 228. DOUBTFUL INFLUENCE OF RACE.

Whether circumstances of *race and nationality* may determine the occurrence of gout, cannot be decided with certainty, for the reason that, in a mixed population such as we always have to consider in the question before us, there are at the same time material differences in the manner of life of the several racial groups; so that it is impossible to be sure what factor has to be credited with the disposition of one race to take the disease, or to what the immunity of another race is to be ascribed. It appears from the statements of Quarrier² and Chassaniol,³ that gout has been seen among the negroes; and if the authorities are not in error it occurs also among Malays in the East Indies, and in China, and among the Hovas in Madagascar. On the other hand we find no references to cases of gout among the Hindus or among the native Indians of North and South America;⁴ but the ques-

¹ I may mention, as a curiosity, that even gout has not escaped the fate of being classed among the communicable (contagious) diseases. Boerhaave was the first to express an opinion to that effect; and such was the great influence of that excellent physician on his contemporaries, that he found many to believe him. Van Swieten, in his commentaries on the 'Aphorisms' (§ 1255, iv, 299), does not altogether deny the contagiousness of the malady, and he adduces in evidence of it the fact that wives who had tended their gouty husbands day and night were ultimately attacked by the disease. But he does not appear to take the matter altogether seriously; at all events he is careful to add: "Licet et multæ aliæ, quæ eodem officio strenue perfungebantur, immunes manserint."

² 'Edin. Med. and Surg. Journ.,' 1808, Oct., p. 459.

³ 'Arch. de méd. nav.,' l. c.

⁴ Schwarz ('Zeitschr. der Wiener Aerzte,' 1858, p. 579) mentions his having

tion that arises here is whether the reason of that exemption is not to be looked for in the mode of life much rather than in the physiological type of the race.

I shall have an opportunity of discussing the *relations between gout and gravel as regards local distribution*, when I come to speak of the latter morbid condition.

been assured by Dr. Candido, who had travelled through Brazil, that he had not met with a single case of gout among the Indians of the country.



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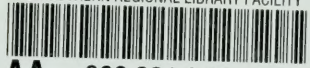
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